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ON

POISONS.

By the same Author,

MEDICAL JURISPRUDENCE.

REVISED BY R. E. GRIFFITH, M. D.

ONE VOL., OCTAVO.

ON

POISONS,

IN RELATION TO

MEDICAL JURISPRUDENCE

AND

MEDICINE.

BY

ALFRED S. TAYLOR, F. R. S.

LECTURER ON MEDICAL JURISPRUDENCE AND CHEMISTRY IN GUY'S HOSPITAL, AND AUTHOR OF "MEDICAL JURISPRUDENCE."

EDITED WITH NOTES AND ADDITIONS,

BY

R. EGLESFELD GRIFFITH, M. D., &c.

> "La justice est armée d'une balance; qu'elle y pèse la parole du savant comme celle du témoin pour les faire servir, l'une par l'autre, à la manifestation de la vérité."
> FLANDIN.

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PHILADELPHIA:
LEA & BLANCHARD.
1848.

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PREFACE.

In laying the present work before the profession, the desire of the author has been to give a more extended view of the subject of Toxicology than he has hitherto been able to introduce into his Manual of Medical Jurisprudence. The crime of poisoning has been of late so fearlessly on the increase, that it seems essential for the proper administration of justice, and for the security of society, to collect and arrange in a convenient form for reference, those important medical facts in relation to death from poison, which, while they constitute a safe guide to the barrister and medical practitioner, may prevent the condemnation of the innocent, and insure the conviction of the guilty. So rapid is the advance of science, and to so great an extent is Toxicology influenced by the progress of collateral sciences, that the lapse of even one year renders numerous additions and alterations necessary. In this volume the author has endeavoured to comprise a full consideration of all questions which appeared to him to have any practical bearing; and these are illustrated by numerous cases of great interest, many of which have not before been published. The discoveries that have been made in the physiological, pathological, and chemical departments of the science, including an analysis of the most important medico-legal trials, have been brought down to the latest period.

It has been considered advisable, for the convenience of the student and practitioner, to publish the work in a form corresponding to that of the Manual of Medical Jurisprudence.

If the subject of poisoning be not fully treated in the present volume, it is neither the fault of the publisher nor of the printer. The former has liberally allowed a wide latitude for a much larger space than was originally intended; and the latter, by the use of clear and legible type, has contrived to introduce into a small and portable volume, a quantity of matter equal to one thousand pages of ordinary type in large octavo. For all faults and omissions,—for any apparent prolixity on some occasions,—and for too great conciseness on others,—the author is alone responsible. His work on MEDICAL JURISPRUDENCE has been most favourably received by the members of the Medical and Legal professions:—it is this which has induced him to spare no pains to make the present volume complete; and he trusts he may rely upon the indulgent consideration of his readers, for any errors into which he may have fallen, in treating so large and comprehensive a subject as that of TOXICOLOGY.

Cambridge Place, Regent's Park,
November, 1847.

EDITOR'S PREFACE.

In presenting an American edition of Mr. Taylor's work "on Poisons" to the public, it is deemed unnecessary to speak of its merits or to point out its great usefulness to the members of the professions of Law and Medicine. It may, however, be stated that it is an elaborate epitome of all that is known on the subject of the Poisons, and is amply illustrated with cases, so as to exemplify the relative value attached by juries to the various symptoms induced by the respective articles; and also of the modes of detecting these articles as given by the best authorities.

In preparing the present edition, not the slightest change has been made in the text of the author, except in the correction of a few unimportant typographical inaccuracies—various additions have, however, been made, which are included between brackets and marked with the letter G. Many of them are in substance the same as those appended to the chapters on Poisons in the American edition of "Taylor's Medical Jurisprudence," but the larger proportion are derived from other sources, due credit being given to the author from whom the information has been culled.

Philadelphia, December, 1847.

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CHAPTER I.

TOXICOLOGY—DEFINITION OF THE TERM POISON.—SALINE MEDICINES POISONOUS IN LARGE DOSES—FATAL EFFECTS OF COMMON SALT. IS WHITE HELLEBORE A POISON?—MEANING OF THE WORDS DEADLY POISON—CASES—DEATH FROM COLD LIQUIDS—MECHANICAL IRRITANTS—SPONGE, POUNDED GLASS—ACTION OF BOILING LIQUIDS.

BY TOXICOLOGY (derived from τοξικὸν, *poison*, and λόγος, *discourse*) we are to understand that science which relates to the history and properties of poisons, and of their effects upon the living body. This subject is commonly regarded and treated as a part of Medical Jurisprudence; but the number and importance of the facts connected with poisons which have been accumulated of late years, have justly contributed to raise toxicology to the rank of a distinct science. Probably there is no branch of medicine in which we meet with a larger assemblage of truths ascertained by observation, and combined under one common character. To the physician, the pathologist, and the medical jurist, a knowledge of the subject is of great importance; for cases are continually presenting themselves in which a practical application of the principles of this science is demanded—as, for example, in the treatment of an individual labouring under the effects of poison—in drawing a clear distinction between changes produced in the body by disease and those caused by poison, or finally in aiding the criminal law in detecting and punishing those who have been guilty of the crime of poisoning.

Definition.—A POISON is commonly defined to be a substance which, when administered in small quantity, is capable of acting deleteriously on the body. It is obvious that this definition is too restricted for the purposes of medical jurisprudence. It would, if admitted, exclude a very large class of substances, the poisonous properties of which cannot be disputed; as, for example, the salts of lead, copper, tin, zinc and antimony, which are only poisonous when administered in very large doses. Nitre, it is well known, exerts a poisonous action only in large, while arsenic is poisonous in small doses; but in a medico-legal view, whether a person die from the effects of an ounce of nitre, or of five grains of arsenic, is a matter of little importance. Each substance must be regarded as a poison, differing from the other only in its degree of activity and perhaps in its mode of operation. The result is the same; death is caused by the substance taken, and the *quantity* required to kill cannot therefore be made a ground for distinguishing a poisonous from a non-poisonous substance. If, then, a medical witness be asked, "What is a poison?" he must beware of adopting this common definition, or of confining the term poison to those substances only that operate in *small* doses.

The fact that a poison has been commonly regarded as a substance which produces serious effects when taken in small quantity, has induced many who have adopted this arbitrary view to assert, that certain substances which have actually been known to cause death, are not poisons; and this doctrine has been apparently strengthened by the fact, that were not some such distinction adopted, it would be difficult to separate the class of poisons from bodies which are reputed inert. In answer to this view, it is perhaps sufficient to show, that there is no good reason for assuming this as the distinguishing character of a poison; for it is impossible, even among substances universally admitted to be poisonous, to make any division according to the effects produced by the quantity taken. In relation to the quantity required to operate fatally, the difference is not so great between cream of tartar and oxalic acid as between oxalic acid and strychnia. If we consider nitre and cream of tartar to be poisons, there seems to be no good reason for excluding common salt (the chloride of sodium.) Medical practitioners would scarcely be prepared to admit this last-mentioned substance into the class of poisons; but it is to be observed, that in a very large dose it is capable of acting as a powerful irritant, and of inflaming the mucous membrane of the alimentary canal to the same extent as much smaller doses of other well-known irritants. An instance of *Common salt* having caused death occurred in the north of England in the year 1839. A young lady swallowed, it is supposed, about half a pound of this substance, for the purpose of destroying worms. It was considered to be a harmless substance, according to the common notion; but in the course of about two hours some alarming symptoms made their appearance, and medical assistance was sent for. She was found to be in a state of general paralysis; and although the stomach-pump and other antidotal means were speedily employed, she died in the course of a few hours. After death there were found the post-mortem changes generally indicative of the effects of a violent irritant on the alimentary passages. (Medical Gazette, 1839–40, i. 559.) This case is deserving of attention, not merely from its novelty, but from the evidence which it furnishes of the fallacy of the popular doctrine, that what is taken so freely in small quantities, without mischief, may be taken, with equal impunity, in large doses. In a toxicological view, we do not see how the effects of salt in this case are to be distinguished from the action of the sulphate or acetate of copper; nor how, if we agree to call the latter substances poisons, we can consistently refuse this appellation to the former. It may appear to be a violation of common language, to call the chloride of sodium a poison, but assuredly it would be a greater inconsistency, to refuse to consider it as noxious, merely because it requires to be exhibited in a larger dose than some other irritants.

It is to be observed that this is not a solitary instance of poisoning by salt. Dr. Christison mentions a case which occurred some years since in London, where a man swallowed a pound, and died within twenty-four hours, under all the symptoms of irritant poisoning. The stomach and intestines were found in a high state of inflammation after death. In another case which occurred to this gentleman serious symptoms were produced in a young man by a much smaller dose. In this instance the individual had taken about *two ounces* as an emetic dissolved in a small quantity of water. He was seized with an acute burning pain in the stomach, tenderness in the epigastrium, and great anxiety, without any vomiting until he drank a large quantity of warm water as a remedy. Before Dr. Christison saw him he had vomited freely, but he still suffered severe intermitting pain. (On Poisons, 659.) In one instance, in which about a table-spoonful of salt had been taken by mistake for sugar, there was no vomiting or purging, but great pain in the region of the stomach, which lasted for several days.

There is another substance commonly reputed to be innocent, but which in a large dose may destroy life. This is the sulphate of magnesia, or *Epsom salts*. A trial took place at the Huntingdon Autumn Assizes, 1842, in which two men were indicted for feloniously killing one Daniel Cox, by administering to him a large quantity of Epsom salts dissolved in beer. The deceased was an old man and a confirmed drunkard, and he was in the habit of drinking beer to excess. On the day laid in the indictment, the deceased had drunk several pints of beer which it was afterwards proved, had been drugged with the sulphate of magnesia. He was seized with violent purging, and died within forty-eight hours. On a post-mortem examination the lining membrane of the alimentary canal was found to be inflamed, and there was no doubt that death was owing to the irritant effects of the salt. One of the prisoners was convicted. The quantity of the substance taken in this case could not be ascertained, but there was reason to suppose that the dose was large.

A case is mentioned by Christison in which a boy ten years old had two ounces of the sulphate of magnesia administered to him medicinally by his father, as a remedy for worms. The salt was taken partly dissolved in a teacupful of water, and very soon after it had been swallowed, the boy staggered and became unwell. When seen by a medical man, half an hour afterwards, his pulse was imperceptible, his breathing slow and difficult, the whole frame in a state of great debility, and in ten minutes more, the child died without any other symptom of note, and particularly without any vomiting. (On Poisons, 657.) It is remarkable that in this case there does not appear to have been any purging, and after death no morbid appearance was found in the body. It has been suggested that substances of this kind connect the true poisons with those which are inert in regard to the economy; but they are assuredly to be regarded by the medical jurist as irritant poisons, and as to the dose administered,—it is of little moment in medicine or in law, whether one grain of one substance or one ounce of another substance be taken, provided the fatal effects be clearly traceable to the action of the particular substance on the body. This is the point to which a medical jurist must direct his attention. The alkaline sulphates in large doses have been found to act in a similar way (see SULPHATE OF POTASH.) In Medical Jurisprudence, therefore, it is necessary to look to the noxious effects produced by particular substances on the system, and the adequacy of these substances to cause death under symptoms of poisoning, rather than to the mere quantities in which they may have been taken.

These remarks on the looseness of the common definition of the term poison have been suggested by the fact that medical men have been sometimes severely pressed in cross-examination on trials for certain criminal offences, to state what is strictly a poison and what is not. In charges of attempted poisoning or of attempted abortion by the administration of drugs, it is by no means an indifferent matter for a witness to be able to say what substances are noxious aud what are inert; or to show, how some bodies commonly reputed inert, may under certain conditions act deleteriously on the system. The conviction of a prisoner may actually depend upon the answer returned by a medical witness to a question of this kind. In this point of view a case which was tried at the Norwich Lent Assizes, April 1846, is well deserving of the attention of medical practitioners. A woman named *Whisker* was charged with administering to the prosecutrix a small portion of White Hellebore (Veratrum album) for the purpose of procuring abortion. When the administration had been clearly proved, an objection was taken to the indictment, on the ground that there was no medical evidence to show that Hellebore was a virulent poison. One medical witness is reported to have said, that Hellebore was noxious to the system and produced ill effects, but he knew of no case in which it had produced death. Under these circumstances he thought he was not justified in calling it a poison. Parke B. in summing up, said that that was a poisonous drug which in common parlance was generally understood and taken to be such; and the evidence for the prosecution he thought was sufficiently strong o bring Hellebore within the meaning of the statute. The jury returned a verdict of guilty, and in answer to the judge said that they considered Hellebore to be a poison.

The remarkable circumstance in this case is, that any medical doubt should have been entertained on the subject. Every toxicologist of repute describes White Hellebore as a poison. It is not so active as many of the vegetable narcotico-irritants, but nevertheless it owes its properties to the presence of a most poisonous alkaloid (veratria) which is diffused through the plant, and therefore renders it poisonous. It has already destroyed life.

The reader will perceive from this case, that whether a particular substance be or be not a poison, is a question of fact left for the decision of a jury from the medical evidence given in the case. In general the indictment contains a clause describing the substance as a poison or "*noxious thing*," a form of expression which should always be adopted, in order to prevent the occurrence of these technical objections.

There is another point of view in which this question may require to be considered, namely, What is to be understood by a *deadly* poison? In most indictments for poisoning, it is customary to describe every poison as *deadly*, a form of expression decidedly bad, and calculated to give rise to legal objections. The substance administered might with equal propriety be described as poisonous, or of a destructive nature; but those who draw up indictments are but little informed on such matters, and they can never speak of a poison without describing it as deadly. The following case occurred on the Norfolk Spring Circuit, 1836. Two persons were capitally indicted for having feloniously caused to be administered to the prosecutor, a quantity of a certain "*deadly poison*," called sulphate of copper (blue vitriol,) with intent to murder him. It appeared in evidence that all the parties were servants in a farmer's family, and that it was the duty of one of the prisoners to prepare breakfast for the other servants. On the morning of the day laid in the indictment, the prosecutor observed that the milk which had been prepared for him was very nauseous, and, after having taken a small quantity of it, he laid it aside. He was soon seized with violent vomiting, but under medical assistance he recovered. The residue of the milk was analysed, and was found to contain sulphate of copper. In the defence, the counsel for the prisoners contended that they could not be convicted of the crime charged in the indictment, since, according to all medical experience, the sulphate of copper was not a *deadly* poison. The medical witnesses, of whom there were two, were then required to give their opinions, but they differed on the point. One, a surgeon of some years' standing, considered it to be a deadly poison, although he admitted that so far as his own experience went, he had had no knowledge of its poisonous effects. The other stated that it was not a deadly poison, and that when sold in a shop, the word poison was never attached to the label. The judge considered the case to be one of suspicion rather than of proof, and the prisoners were acquitted. Although, by this summary disposal of the case, the force of the objection to the indictment was rather evaded than decided, yet the difference of opinion between the two medical witnesses is worthy of remark. The question may be easily raised again, and there ought to be some understanding among practitioners as to the proper signification of the word *deadly* when applied to poisons.

It appears to me that the term *deadly* can be used with respect to those poisons only which may prove speedily fatal in small doses, such as strychnia, morphia, prussic acid, and arsenic; and that it could not with any sort of propriety be applied to such a substance as the sulphate of copper. The error essentially lies in the legal wording of the indictment, with which, of course, a medical witness is not concerned. If an objection of this kind is to be held valid, and a question of criminal poisoning to be dismissed on so trivial a point, it is reasonable to expect that greater care should be used in drawing up indictments, as also that medical terms should not be employed by non-medical persons without proper supervision:—otherwise, it is obvious that the ends of justice must be defeated. Differences of opinion among educated medical witnesses, are however not likely to exist where slight reflection has been bestowed upon the subject.

A similar question arose in a trial which took place at Chelmsford some years ago, where the substance administered was copperas or sulphate of iron. A man was charged with having administered this substance to two women, with intent to murder them; and in the indictment it was described as a *deadly* poison. The medical witness stated that it was not, properly speaking, a deadly poison. This is so far true, that it is rare to meet with a case in which this substance has destroyed life. There is no doubt that green vitriol is capable of acting as an irritant, and as such, if taken in a large dose, and not ejected from the stomach by vomiting, it may produce inflammation of the viscera and death. It is clear, however, that a person who described copperas as a deadly poison, could have had but little acquaintance with the subject of poisons. If any advantage is to be taken by prisoners from such a misapplication of medical words in indictments for this crime, it is only just that the preparation of these instruments should be entrusted to persons possessing some knowledge of legal medicine.

After all, it must strike the reader that an objection of this kind is too trivial to be entertained. It would seem reasonable that the proof of the crime of poisoning should rest either upon the fact of the substance administered being a poison, or of its having the power of operating as such. Whether it be strictly of a deadly nature or not, should be considered a matter entirely subordinate to the main inquiry. This question again arose in the case of *Reg.* v. *Haydon*, Somerset Spring Assizes, 1845. The substance administered in this case was "spirits of hartshorn," which was most absurdly described as a "deadly poison." The counsel for the prisoner took an objection to the indictment on this ground, but the judge (Erle, J.) said, "the word *deadly* appears to me to be used merely in pursuance of an ancient form, and not to be essential to the validity of the indictment. It would be sufficient to describe it simply as a poison, and under that term would fall, any thing calculated to destroy life. Substances harmless in themselves might become poisons by the time or manner of their administration. This seems to me the view most accordant with common sense, and therefore I hold this indictment to be good, even though it describes spirits of hartshorn as a *deadly poison*." (Law Times, April 12, 1845.) The question may now, therefore, be considered as settled. The word *deadly* must be regarded as mere surplusage.

In legal medicine, it is difficult to give such a definition of a poison as shall be entirely free from objection. Perhaps the most comprehensive definition which can be suggested is this: "A poison is a substance which, when taken internally, is capable of destroying life without acting mechanically on the system." Some substances may, however, act as poisons by absorption when applied to the skin or a wounded surface; (see CORROSIVE SUBLIMATE, CANTHARIDES, and ARSENIC,) while others again, as the poison of the viper, and of hydrophobia, may have their fatal effects limited to those cases in which they are introduced by a wound. Leclerc, who wrote in 1803, defines a poison to be a substance which, when taken in a small dose internally, or applied to the living body in any way externally, is capable of injuring health or causing death (Essai Médico-légale sur l'Empoisonnement, p. 49, Paris, An. xi.) This definition has been substantially adopted by some modern toxicologists. It is, however, not only open to the objection that most poisons must be taken in *large* doses in order to injure health or cause death, but it would include melted lead or boiling liquids among poisons; for these, when applied to the living body externally, injure health and cause death. M. Flandin, one of the most recent writers on toxicology, considers only those substances to be poisonous, which are capable of entering into the body by absorption, and which produce dangerous effects only when absorbed (Traité des Poisons, i. p. 193.) This definition is inadmissible, because it would obviously exclude from the class of poisons the mineral acids and alkalies. A similar objection appears to me to exist to the definition of a poison by Dr. Griffith, the American editor of my Manual of Medical Jurisprudence. "A poison is a substance which, when taken internally, or applied to the surface of the body, is capable of destroying life without acting in a purely mechanical manner." Can it be said that boiling water or oil, or melted lead, when applied to the surface of the body, destroy life by exercising a mechanical action? If not, then these liquids are poisonous. If, however, this kind of action be regarded as mechanical, then the mineral acids and alkalies must also be considered by their effects to act mechanically, whether applied to the skin or the mucous membrane of the stomach.

These remarks show that it is, indeed, very difficult to comprise in a few words an accurate description of what should be understood by the term "poison."

Under the definition which I have above given, it might be objected that the whole class of medicines, and numerous substances of an inert nature, would be included. Thus it is well known, that there are many cases on record in which *cold water*, swallowed in large quantity, and in an excited state of the system, has led to the destruction of life either rapidly by shock, or slowly by inducing gastritis. Any cold liquid, such as iced water, beer, or ice itself, may have an equally fatal effect. The action of water or cold liquids, under these circumstances, cannot be said to be mechanical; it appears to be due to the shock suddenly induced on the nervous system through the lining membrane of the stomach, and yet it would be inconsistent to class these inert liquids among poisons. In regard to the effects of cold liquids and the medico-legal questions which arise respecting them, see an elaborate paper on the subject, by Dr. Guérard, in the Annales d'Hygiène, 1842, i. 42.

In all cases of this description, it appears to me that we are justified in drawing the following distinction between poisonous and non-poisonous substances. If the deleterious effect does not depend upon the nature of the substance taken, but upon the state of the system at the time at which it is swallowed, the substance cannot be regarded as a poison. All poisonous substances are *per se* deleterious,—the state of the system, setting aside for the present the peculiar effects of idiosyncrasy and habit, has very little influence on their operation. The symptoms may be suspended for a time or slightly modified in their progress, but sooner or later the poison will affect the healthy and diseased, the old and the young, with a uniformity in its effects not to be easily mistaken. A distinction of this kind cannot, however, be drawn, except by a professional man, who has given attention to the subject of toxicology; and therefore it is no matter of surprise that poisoning should have been in more than one instance erroneously imputed, in cases where death has followed the drinking of cold liquids.

In thus giving the *medical* definition of a poison, it is necessary to observe that the law never regards the manner in which the substance administered acts. If it be capable of injuring the health of an individual, it is of little consequence, so far as the responsibility of a prisoner is concerned, whether its action on the body be of a mechanical or chemical nature. Thus a substance which simply acts mechanically on the stomach, may, if wilfully administered with intent to injure, involve a person in a criminal charge, as much as if he had administered arsenic or any of the ordinary poisons. It is then necessary that we should consider what the law strictly means by the act of poisoning. If the substance criminally administered destroy life, whatever may be its nature or mode of operation, the accused is tried on a charge of murder, or manslaughter, and the whole duty of the medical witness consists in showing that the substance taken was the certain cause of death.

If, however, death be not a consequence, then the accused is tried under a particular statute for the attempt to murder by poison (1 Vict. c. lxxxv. sec. 2.) The words of this statute are very general, and embrace all kinds of substances, whether they be popularly or professionally regarded as poisons or not. Thus it is laid down that "Whosoever shall administer or cause to be taken by any person, any poison, or other destructive thing, with intent to commit murder, shall be guilty of felony, and being convicted thereof, shall suffer death." Whether the administering be followed by death or bodily injury dangerous to life, it is still a capital felony provided the *intent* have been to commit murder. The same administering with intent, &c., although no bodily injury be effected, is felony, punishable by transportation for life, for fifteen years, or imprisonment for any term not exceeding three years. From the words of the statute it appears that the law requires, in order to constitute the crime of poisoning, that the substance should be *administered to*, or be *taken by*, an individual. These words do not appear to be sufficiently comprehensive. Several deaths have been caused of late years by the external application of arsenic and corrosive sublimate to ulcerated and diseased surfaces. Supposing that poison is thus applied intentionally, and great bodily injury is done to the individual, it is open to doubt whether the crime would be punishable under these sections of the statute.

MECHANICAL IRRITANTS.—Such, however, is the present state of the law of England in respect to attempts at poisoning when death does not take place. While the words of the statute render it unnecessary for a medical witness, in such cases, to give judicially a very close definition of "a poison," they impose upon him a difficulty which he must be prepared to meet. The substance administered may not be a poison in the medical signification of the term, nor may it be popularly considered as such; and yet, when taken, it may be destructive to life. We have examples of substances of this description in iron filings, powdered glass, pins and needles, and such like bodies, all of which have been administered with the wilful design of injuring, and have on various occasions given rise to criminal charges. In cases of this kind, the legal guilt of a prisoner may often depend on the meaning assigned by a medical witness to the words *destructive thing*. Thus, to take an example,—liquid mercury might be poured down the throat of a young infant, with the deliberate intent to destroy it. A question of a purely medical nature will then arise whether mercury be "a destructive thing" or not; and the conviction of the prisoner will probably depend on the answer returned by the witnesses. Should a difference of opinion exist,—an occurrence by no means unusual in medical evidence, the prisoner will, according to the humane principle of our law, receive the benefit of the doubt. The point which here requires to be considered is, why any difference of opinion should exist among witnesses.

With regard to the case just supposed, it is a general principle in toxicology, that the pure metals are not poisonous; and they are not to be regarded as "things" destructive to life, unless the mechanical form in which they are taken be such as to injure the viscera with which they may come in contact, leading to inflammation and its consequences. Even where the mechanical form favours the production of these serious results, especially perforation of the intestines, the powers of nature are often exerted in a most extraordinary way, and the individual lives apparently in good health. This has been witnessed in the cases of many who have swallowed knives, or pins and needles. The escape of such persons must, however, be regarded as the result of accident. They are always in imminent danger, and they commonly die sooner or later from inflammation or perforation of the viscera. For a remarkable case in which death took place from this cause in Guy's Hospital, see Medical and Physical Journal, October, 1809, p. 350. The masses of iron which caused death in this case are preserved in the Museum collection. For another interesting case in which death took place from disease of the brain, and a large number of iron nails and other metallic substances were found in the stomach, see Dublin Medical Journal, September, 1835; also Med. Gaz. xvi. 791.

Liquid *mercury*, the substance which we have here taken as an illustration, cannot operate deleteriously on the body either chemically or mechanically. It may be taken, and has often been swallowed in very considerable quantities, without perceptibly affecting the health. If a medical witness were not aware of these facts, and did not sufficiently reflect upon the nature of the question addressed to him, he might improperly cause the conviction of an accused party. The intention of the accused may have been criminal, but that is a matter unconnected with the duties of a witness:—he is simply required to state whether the means employed to carry out this criminal intention, were such as were likely to produce danger to life. Similar observations might be made with regard to numerous other substances employed in medicine or in the arts; and it is quite obvious that difficulties of this kind can only be properly met by those practitioners who have closely attended to the subject of toxicology. It is well known that bodies which are not in their own nature destructive, may become so through indirect causes. Metallic iron is not a poison, nor can it, except under certain circumstances, be regarded as a destructive thing. An angular mass of iron may, however, kill by perforating the viscera; or, if the metal be exhibited in the state of filings, in large doses frequently repeated, then it may become a source of irritation in the stomach, and lead to ulceration and perforation. There will be no difficulty, however, for a practitioner to form an opinion in this and all similar cases. Sometimes the substance may be of a nature to produce a poisonous compound in the alimentary canal. Metallic arsenic is not considered to be poisonous, but it is capable of forming arsenious acid when in a finely-divided state, and thus leading to death. Thus metallic arsenic, although not a poison, must still be regarded as a "destructive thing." The metal copper may act on the system either mechanically or chemically: when in the alimentary canal it may cause death by ulceration, or it may produce, with the acid and mucous liquids, the subchloride, acetate, or carbonate of copper. Sometimes the metal may acquire a coating of sulphuret, in which case its operation would be mechanical, since the sulphuret of copper is not poisonous. The rapidly destructive effects of these metallic substances, when acting mechanically, are well shown by a case reported by Mr. Dicken (Med. Gaz. xxxv. 885.) A boy, aged ten years, accidentally swallowed a copper halfpenny. There was very slight constitutional irritation: purgatives were administered, but the coin was not passed. There was merely a sense of weight and uneasiness in the epigastrium, with a feeling of distention, which subsided in a few hours. On the twenty-seventh day after swallowing the coin, he was suddenly seized with sickness, and instantly vomited more than a quart of arterial blood. The bleeding continued the following day, when he felt something pass from his stomach into the bowels—the hæmorrhage recurred, and he died on the twenty-ninth day. On opening his stomach, a circular patch of ulceration was observed on the mucous membrane, at a considerable distance from the pyloric orifice: but there was no appearance of inflammation. The coin was found at the termination of the large intestines, lying loose and easily removable. There was no morbid appearance on any part of the alimentary canal, except the circular ulcer in the stomach, probably caused by the coin which had led to death by hæmorrhage. The coin had not undergone any chemical change. Gold, silver, and tin have been occasionally swallowed. These metals can only act mechanically.

Among the singular methods resorted to for the purpose of destroying the lives of infants and children, that of causing them to swallow *pins* or *needles* in their food, is one which claims the attention of medical jurists. This mode of perpetrating murder has been brought to light by the evidence given on several criminal trials, which have taken place of late years in England and on the continent. In cases of this kind, death is commonly to be referred to inflammation: and a practitioner can have no hesitation in designating these bodies, when exhibited to young children, as "destructive things:" they are at all times likely to lead to serious injury, if not to death; nor is it any answer to this view to assert, that they have been often swallowed with impunity. We know that active poisons are sometimes taken without causing death; but this does not alter our opinion, that they are substances destructive to life, and likely to give rise to the most serious consequences. A case is reported in the Medical Gazette (vol. xxvi. p. 582) which will show how far the powers of nature are sometimes capable of resisting the effects of these mechanical irritants. Here it appears that two hundred and fifty-four pins and needles were removed from a female aged 23, in whose body the greater number had remained for a period of thirteen years. An interesting case is reported by Dr. Neumann, in which a pin accidentally swallowed remained in the body of a young woman for a year, and was finally discharged through the skin without injurious consequences. (Casper's Wochenschrift, 1846, 180.) That death may ensue from this cause, is an undoubted fact. In the Registrar's report for 1838–9, one child is stated to have died from the effects produced by swallowing a pin. In August 1841, a boy aged eleven years was committed to Newgate on a charge of theft. Shortly after his imprisonment, he swallowed a quantity of pins for a trifling wager. He soon afterwards became extremely ill, and died in the course of a few weeks, evidently from the effects of the mechanical irritant which he had swallowed. A girl was tried in France, in 1838, upon a charge of having endangered the life of an infant aged five weeks, by administering to it pins. The medico-legal investigation of this case was entrusted to M. Ollivier. From the evidence given on that occasion, it appears that in the opinion of M. Ollivier, these mechanical irritants are likely to produce more serious effects in an adult than in an infant; but this view is not based upon any particular facts. He also asserted that pins and needles, when swallowed, were comparatively harmless, and that a fatal termination was the exception to the rule. (Annales d'Hygiène, t. xxi. 178.)

In regard to the opinion expressed by M. Ollivier of the non-production of serious consequences by these mechanical irritants, it may be observed that there is a sufficient number of fatal cases on record to justify us in asserting that they are destructive things, and may endanger life. The following is, in this respect, a case of some interest, since, on a post-mortem examination, death was clearly referrible to the mechanical irritant. A female was charged with having caused the death of a child by administering to it pins. Nine days after its birth, a pin was found in its mouth; and in about six days afterwards the child died. The mother confessed that she had caused it to swallow pins for the purpose of destroying it. The child had been born prematurely, and was of weakly habit. The abdomen was carefully examined, and the transverse arch of the colon, with the small intestines near it, were in a state of inflammation. On turning over the liver, it was found to be penetrated on its under surface near the gall-bladder by a pin, the head of which could be felt within the duodenum. The liver, pancreas, and intestines were glued together by bands of adhesive matter. On laying open the cavities of the viscera, the head of the pin was found to be near the pylorus. The pin was of a large or coarse kind, about an inch and a half in length; and after traversing the parietes of the duodenum, it had penetrated into the liver for about an inch, from before backwards. There were marks of inflammation in the liver, but the other viscera were healthy. Death was undoubtedly due to the inflammation produced by this mechanical irritant. (Henke, Zeitschrift der S. A. 1838.)

It is obvious that the fatal effects thus produced by pins or needles must be in a great measure accidental. It is not from the number of these articles swallowed, nor from the age of the subject, that any just inference can be drawn as to the probability of their proving fatal to life. If it be true, as M. Ollivier has asserted, that death is the exception to the rule, it is not the less true, that the life of any individual who has swallowed pins or needles, is always in danger until they are discharged. Sometimes, in these cases, life may be suddenly destroyed by hæmorrhage. Mr. Bell has published a case in which a young man aged 18 accidentally swallowed a needle in soup. In the course of ten days, he had several attacks of spitting of blood, and in one of these fits he vomited a large quantity, and expired in a few minutes. On examination, a fine sewing needle was found lying across the œsophagus, the point of which had penetrated the right common carotid artery, and had led to the fatal hæmorrhage. (Med. Gaz. xxxi. 694.) For a very ingenious method of detecting needles in the body when buried beneath the skin, I must refer to a pamphlet by Mr. A. Smee, London, 1845. That a medical jurist must be prepared for giving evidence on the effects of mechanical irritants will be proved by the following case, which came to a trial on a charge of murder at the Chelmsford Assizes in 1835. The prisoner, an old woman, was indicted for the murder of her grand-daughter, by causing her to swallow some sponge and a piece of wood. It was also suspected that she had administered pins to the child. The deceased was eleven weeks old; and until within a very short period of its death, it had appeared to enjoy very good health. The evidence of the only medical witness examined, was to the following effect. He stated that in a post-mortem examination of the body of the child, he had found the mucous membrane of the stomach, as well as the liver, inflamed, and there were adhesions of the peritoneum. The stomach contained a piece of wood, and there were several pieces of sponge in the large intestines. On inspecting the viscera more closely, he discovered a pin in the substance of the liver, on its convex surface next the stomach. The pin was discoloured by the fluids of the body. The substances which he found in the body were sufficient to produce inflammation; and it was, in his judgment, this inflammation that had caused the death of the child. The witness could give no opinion as to how the pin had penetrated into the liver. On cross-examination, he admitted that the pin might have found its way into the cavity of the abdomen by accident. The wood and sponge might also have been accidentally introduced during the dressing and feeding of the child. It was left to the jury to say, whether the substance found in the viscera, and which, by mechanical irritation, had led to inflammation and death, had been introduced wilfully or accidentally, and as there was no direct evidence on this point, they acquitted the prisoner.

In this case the mechanical irritation was probably as much due to the sponge as to the pin. The quantity of sponge found within the intestines was small. It is difficult to conceive how these different substances could have been accidentally swallowed by an infant.

Sponge may be regarded as a mechanical irritant; but little is known concerning its action on the human body. In the Medical Gazette (vol. xxxi. p. 124,) two cases are related in which this substance was swallowed by a horse. In one case, it did not appear that the animal suffered any inconvenience; but in the other case, it became alarmingly ill. There can be but little doubt, that where sponge in large quantity remains lodged in the viscera, it is capable of producing inflammation and death. Dr. Chowne has, however, lately reported a case in which a small piece of sponge accidentally swallowed by an infant, produced no injurious effect.

Certain articles of food may even act mechanically and destroy life, by simple over-distention of the organ. A singular instance of this kind, in which a quantity of *raw rice* was the immediate cause of death, occurred in December 1846. A young woman, æt. 22, ate a tumbler-full of raw rice mixed with milk, just before taking her tea. In a few hours she became suddenly ill with severe pain in the region of the stomach and great restlessness, evidently due to the distention of the organ from the swelling of the rice by the imbibition of the fluid. Emetics were given with great relief, a large quantity of rice being expelled from the stomach. The next morning the pain increased suddenly and violently, with cold extremities, small feeble pulse, and great abdominal tenderness. She died about twenty-four hours after taking the rice. On examination the peritoneum was found extensively inflamed; there was a deposition of lymph, with a copious effusion of turbid serum. The stomach and duodenum were empty and free from inflammation. (Case reported by Mr. Howell, Lancet, April 10, 1847.

A singular case was referred to me in April, 1847, in which there was great reason to believe that a child, aged seven years, had died from the effects of a quantity of *orange peel* with which it had gorged its stomach. It complained of severe pain in the stomach, vomited repeatedly a yellowish coloured matter, became convulsed, and died in twenty-four hours. A quantity of yellow fluid with orange pips and orange peel was found in the stomach. The mucous membrane was generally reddened, and in several places there was well-marked injection. No poison was found, nor was there reason to suspect, except from sudden death, that any had been administered. There was no other cause for death but the irritant effects of the large quantity of orange peel eaten by the child.

Among mechanical irritants, there yet remains to be mentioned one, which was formerly regarded as an active poison, namely, *pounded glass*. Recent observations have satisfactorily shown, that this substance is not a poison. It is liable to inflict injury upon the alimentary canal, just in proportion to the size and sharpness of the fragments; and whenever it is swallowed in a state of coarse powder, it may irritate and excite inflammation of the stomach and bowels. Glass, in very fine powder, is decidedly alkaline; but it does not possess any of the properties of an alkaline poison:—on the contrary, in that condition in which its alkalinity is most manifested, it appears to be inert. It is said, that six or seven ounces of this substance have been given to a dog without producing any inconvenience to the animal. A trial for murder occurred in Paris in 1808, in which the accused was charged with having poisoned his wife by administering to her pounded glass. This substance was found in the stomach, and both this organ and the intestines exhibited marks of great irritation. Baudelocque and Chaussier gave their opinion that the glass was not the cause of death. Portal relates an instance of a young man who during a debauch broke a glass between his teeth, and then swallowed some of the fragments. These were afterwards expelled by active vomiting, and he recovered. In an attempt made by an ignorant person some years since to poison a whole family with coarsely powdered bottle-glass mixed with food,—no inconvenience resulted to those who had swallowed a portion of the glass. A case is, however, reported, in which it appears highly probable that a child aged eleven months was killed by the administration of this substance. Powdered glass was found in the mucus of the stomach, and the lining membrane itself was very vascular. (Christison, 654.) It is obvious that a substance of this description cannot be easily swallowed by an adult without his being perfectly aware of it; and the instances in which it has been administered to infants are very few in number. Although I believe the only instance reported of its having acted fatally is in the case of the infant just described, yet a medical jurist cannot hesitate to say that pounded glass is a mechanical irritant; and that the irritation caused by the presence of a large quantity of this substance in the stomach or bowels, might lead to fatal gastritis or enteritis. For a highly interesting case, in which a medico-legal question was raised on the effects of pounded glass and mechanical irritants generally, see Annales d'Hyg., 1830, i. 364. Experiments on this subject have been recently performed by Dr. Rufz. (Annales d'Hyg., 1844, ii. 195.)

Boiling Liquids.—Some toxicologists have placed hot liquids, such as *boiling water* or *oil*, in the class of mechanical irritants; but the effects produced by such liquids cannot with propriety be said to be mechanical. They do not act like poisons, although they leave in the body changes similar to those produced by corrosive poisons. Death from accidental swallowing of boiling water is by no means uncommon among young children. According to the observations of Dr. Hall and Mr. Ryland, the fatal result is most commonly to be ascribed to inflammation of the fauces and larynx, produced by the contact of the boiling liquid. This inevitably leads to suffocation, unless assistance be at hand. In a case of recent occurrence, a child was actually asphyxiated from this cause, when my friend, Mr. E. Cock, by the timely performance of tracheotomy and inflation of the lungs, succeeded in restoring it. Sometimes, however, inflammation of the stomach is a consequence. A case of this kind occurred a few years since at Guy's Hospital, and on a post-mortem examination, the mucous membrane at the larger end of the stomach was found to be much inflamed. The appearance was very like that produced by the common mineral irritants, although it was more confined to one part of the mucous membrane. In the Registrar's report for 1838–9, twenty-four deaths are stated to have occurred among young children from this cause alone!

CHAPTER II.

MODE IN WHICH POISONS OPERATE—LOCAL ACTION—REMOTE ACTION—PERIOD REQUIRED FOR THE ABSORPTION AND ELIMINATION OF POISONS—VISCERA IN WHICH POISONS ARE FOUND—ELIMINATION OF ARSENIC—DETECTED IN THE FŒTUS IN UTERO—RAPID ABSORPTION—ACTION BY SYMPATHY—CAUSE OF DEATH—HABIT—IDIOSYNCRASY.

In investigating the phenomena which attend the operation of poisons, we are led to inquire into the mode in which they affect the body and cause death. This inquiry is highly interesting in a physiological and pathological view; but it is not of much importance to a medical jurist. In the generality of cases, all that the law requires to be established by medical evidence, is that the substance taken was adequate to cause death. Nevertheless, in a recent case of poisoning by opium, a medical witness was specially examined by the Court as to the mode in which the drug was supposed to cause death; and in other instances, questions of a similar import have been put with respect to other poisons. This is sufficient to justify the introduction of a few remarks on the subject in this place.

Poisons have either a *local* or *remote* action upon the system; and in the greater number of cases, both of these actions are manifested by the same substance.

LOCAL ACTION.—The local action of poisons is most strikingly seen in those substances which are of a corrosive nature, such as the mineral acids and alkalies. A chemical change is induced by these agents in the structure of the part with which they come in contact, whether it be on the outside or inside of the body; and should the disorganization produced be very extensive, death will take place as in any other case of mechanical lesion to a vital organ. If the individual survive the first effects, and the poison be not neutralized or removed from the stomach, the local irritation produced may give rise to inflammation, with ulceration, gangrene, and their consequences. But the local action of a poison is not always indicated by physical changes in a part. The effect may be confined to the sentient extremities of the nerves only, manifested by the occurrence of paralysis. It is well known that aconite, morphia, and prussic acid are capable of affecting the nerves, if they remain sufficiently long in contact with a part: and many experiments have proved, that the nerves supplying the hollow viscera, through which sensation is not manifested, are equally susceptible of this local action. Opium applied directly to the intestines has been known to put an end to their peristaltic motion; and the same phenomenon was accidentally observed by Addison and Morgan in their experiments with the ticunas poison. From these facts it has been inferred, that certain poisons may act in a similar way upon the stomach, or upon the nervous fibres of the part to which they are applied, and a fatal impression be transmitted to the brain or spinal marrow. It must be admitted, however, that there are no conclusive experiments to show that the impression indicated by paralysis of motion or sensation is actually conveyed through the nerves. On the contrary, some experiments lead to the inference, that the impression is essentially local, or restricted to the part touched by the poison. The numbness produced by prussic acid on the skin is confined to that part only to which the acid is directly applied: it does not appear to extend in any perceptible degree beyond this part. The experiments lately performed by M. Serres with liquid ether have furnished similar results. Having laid bare the nerves of the thighs of several rabbits, and placed them in contact with a sponge dipped in ether, it was observed that the sensibility of the nerve was destroyed at the points immediately in contact with the ether, but the entire sense of feeling remained in the portion of nerve above the point immersed in ether. In order to determine how far exposure to air affected the results, two nerves were laid bare; one was immersed in ether, and the other simply exposed to the air: in five minutes the nerve in ether was dead to all sensation, even on the application of pincers, while the nerve exposed to air retained all its sensibility and power of exciting contraction. The application of tincture of nux vomica, strychnia, or its salts, did not produce the least sign of sensibility or power of contraction (Academy of Sciences, February 8, 1847.)

The difference between the local action indicated by physical change, and that which is unaccompanied by any such change, is this:—that in general the former, being chemical, takes place equally in the dead and the living—the latter, of course, in the living subject only.

There are certain poisons, concerning the local action of which some doubt exists among toxicologists. Thus arsenic possesses no corrosive action; it does not chemically destroy a part; and, although we might infer from the extensive morbid changes which are observed in the stomach in cases of arsenical poisoning, that it must have a powerfully local chemical action, yet there are many facts which are strongly opposed to the admission of this view. Thus, inflammation of the stomach has been found in cases where the arsenic was applied externally to a wound or an ulcer. Again, we do not find that the degree of inflammation is in proportion to the quantity of the poison taken; sometimes it is extensive under a small dose, and at others scarcely apparent under a large dose. When arsenic, in powder or solution, is placed on dead mucous membrane, no chemical change whatever takes place; and an examination of the mucous membrane of the stomach of a person poisoned by it, is commonly sufficient to show that it has no local action like the corrosive poisons. Yet it cannot be denied, that it most strongly irritates, although it does not chemically destroy the delicate lining membrane of the viscera.

In cantharides, we have a substance which acts locally by irritating and inflaming the part to which it is applied, whether this be the stomach or the skin. It neither chemically corrodes the animal substance, nor does it appear to have any paralyzing action on the nerves. The local action of a corrosive poison is often changed by combination. Thus, pure barytes, from its causticity, exerts a chemical action on the stomach and acts as a corrosive; but when combined with carbonic acid, under the form of carbonate of barytes, it acts simply as an irritant. Hence it would appear that poisons may operate locally in three ways:—1. By chemically destroying the part with which they come in contact. 2. By paralyzing the sentient extremities of the nerves. 3. By simply irritating the part, and giving rise to inflammation and its consequences.

Remote action.—By this we are to understand that power which most poisons possess of affecting an organ remote from the part to which they are applied. The same substance often possesses both a local and remote action: but some poisons affect one organ remotely, and others, another. Cantharides, a poison which has a violent local action as an irritant, to whatever part of the body it may be applied, affects remotely the urinary and generative organs. Mercury affects the salivary glands. Morphia, whether applied to a wound or to the mucous membrane of the stomach, affects the brain. Digitalis taken internally affects the heart; strychnia, the spinal marrow. In some cases, this kind of action is more obscure; and the same poisons will affect remote organs differently, according to the form and quantity in which they may have been taken; and, perhaps, according to peculiarity of constitution in the poisoned subject. The mineral acids rarely affect the brain remotely: the mental faculties, in cases of poisoning by them, commonly continue clear until the last moment of life. Arsenic sometimes affects the heart, which is indicated by syncope;—at other times the brain and nervous system, which is known by the coma, stupor, and paralysis that occasionally supervene in poisoning by this substance. Oxalic acid has been found by Christison and Coindet to affect remotely either the heart, the spinal marrow, or the brain, according to the strength of the solution in which it was administered to animals.

In all cases of acute poisoning, whether the substance have a local action or not, death must commonly be ascribed to the influence exerted on a remote organ important to life. Most poisons destroy life by affecting the heart, the brain, or spinal marrow. The impression produced on either of these important organs is not always so intense as to kill; for individuals have been known to recover even when alarming symptoms from this remote influence had manifested themselves. In some instances, however, the impression produced is such as to annihilate speedily, the vital functions. Thus, large doses of hydrocyanic acid or strychnia kill with great rapidity, without producing any perceptible local changes. Of late years, it has been stated, that hydrocyanic acid possesses an irritant action; but this is by no means a condition necessary to its fatal operation as a poison. Even when local changes of any extent are met with, as in acute poisoning by the mineral acids, death is still to be ascribed to a fatal impression produced on a remote organ,—commonly the heart. Whatever gives rise to similar lesions in the stomach, whether the cause be chemical or mechanical, *e. g.* boiling water, will operate in like manner. In the action of the mineral acids, the fatal effect is not commonly so rapid, as where there has been a destruction of the organ from causes of a mechanical nature. A person has been known to fall dead in a few seconds from a lacerated wound of the stomach produced by a pistol bullet, and there has been no hæmorrhage to account for this rapid death; while in poisoning by sulphuric acid, when the coats of the viscus have been extensively perforated, and the acid has escaped into the abdomen, some hours have elapsed before the individual has died. Thus, then, poisons which have the most extensive local action, kill by affecting remote organs important to life, just like mechanical injuries of similar extent. There may be a difference in the time at which death takes place; but the cause of death is the same, namely—a remote operation by sympathy, otherwise called shock.

Nothing is more common than to hear it said in cases of arsenical poisoning, that the local changes are sufficient to account for death. These changes, which are due to the irritant properties of the poison, should, however, be regarded rather as accompaniments of its action, than as absolutely necessary to explain its fatal effects; although it cannot be denied, that violent inflammation, attended by ulceration or gangrene, may suffice to account for death, as in cases of severe gastritis produced by any cause whatever. In this and in most other instances, when the substance is simply irritant, death is commonly due to the remote influence of the poison. This view appears probable from the fact, that if arsenic be removed from the stomach before it has had time to produce any well-marked local changes, the case may nevertheless prove fatal by the effect of that portion which has been absorbed and carried into the circulation. Again, it is well known, that three or four grains of arsenic, a quantity insufficient to produce any striking local changes, will destroy a person under all the usual symptoms of poisoning by this substance. The same may be said of corrosive sublimate:—three or four grains of this poison would suffice to kill an adult; and yet from this small quantity, the local changes would be barely perceptible.

Thus, then, with regard to poisons generally, it is established that whether they chemically corrode, irritate, or produce no apparent alteration in the part to which they are applied, they destroy life by producing a fatal impression upon a remote vital organ. That death should ever take place in poisoning without any physical changes being produced on the body, is not more wonderful than that it should occur under attacks of tetanus or hydrophobia, in which diseases, as it is well known, no post-mortem appearances are met with sufficient to account for their rapidly fatal course.

Two questions here present themselves for our consideration: 1. In what way is this remote influence of poisons conveyed to the vital organs? and 2. How does it act on the vital organs to destroy life?

With regard to the solution of the first question, there is still great difference of opinion among toxicologists. It may be sufficient to say, that all are agreed that the influence must be conveyed either by the blood-vessels or by the nerves. According to the first view, *absorption* is necessary to the action of a poison; and according to the second, all that is requisite is, that the poison should come in contact with the nervous filaments of any part of the body. In the last case, some have considered that the influence is conveyed by the cerebro-spinal, and others by the ganglionic, system of nerves. It would be out of place to enter into the respective merits of these theories; it will be merely necessary to state a few of the facts which have been derived from experiments on the subject.

ABSORPTION.—We shall first inquire whether poisons enter into the blood: and if so, whether the entrance into that fluid is absolutely necessary to the production of their fatal effects. It has been long known that certain mineral substances, when taken internally or applied externally to a wound, can be detected in the blood, and most of the secretions of the body. Among these the acetate of lead, sulphate of iron, and chloride of barium, have been found in the blood; and it is well known that the iodide of potassium can be readily detected in the urine of persons who are taking it medicinally. The following experiment was performed many years since by Mr. Key:—a quantity of ferrocyanide of potassium in powder was rubbed into a wound on the inside of the leg of a donkey; and about six hours afterwards the animal was killed. Mr. Key forwarded to me for analysis one portion of blood taken from the femoral vein, another portion from the mesenteric veins, and lastly the contents of the thoracic duct. The ferrocyanide was readily detected in the three specimens, being most abundant in the blood of the femoral vein, and least abundant in the contents of the thoracic duct. From experiments similar to this, it has been inferred that most poisons enter into the circulation.

But an inference of this kind does not rest upon bare analogy. Within the last few years arsenic has been detected in the blood of persons poisoned by arsenious acid, both during life, and after death. It mattered not from what part of the body the blood was taken, arsenic was equally discovered; so that from these and other experiments, it would appear that the living or dead body in a case of arsenical poisoning, is for a time penetrated throughout by the poison, and during life it appears to be constantly eliminated in the urine and other secretions. The fact that arsenic may be detected in the blood and urine of a person who survives its effects, is a point of considerable importance in a medico-legal view. Thus an analysis of either of these fluids may furnish evidence otherwise only satisfactorily obtained by a post-mortem examination of the body; and cases of the criminal administration of arsenic to the living, which have hitherto escaped the hands of justice, owing to the want of chemical proof, may become as clearly established to the satisfaction of a jury, as if the poison had operated fatally and had been found after death in the stomach. Arsenic has been repeatedly discovered by toxicologists in the viscera of those who have been poisoned by it, even after the bodies have been interred for many years. Antimony has been detected by Orfila in the urine of persons to whom tartar emetic was administered, and also in the substance of the viscera of animals killed by it. It was not discovered by him either in the blood or any of the liquids of the body except the urine. Copper was found by Orfila in the substance of the viscera of animals to which the poisonous salts of this metal were given, but not in the blood or secretions. Tiedemann and Gmelin discovered verdigris in the venous blood of horses poisoned by it; and the same chemists detected acetate of lead under similar circumstances. In an accident which occurred to a cow, where the animal swallowed a quantity of carbonate of lead mixed for paint, I detected traces of lead in the milk drawn some hours after the poison had been taken. M. de Kramer, of Milan, has announced that he has detected nitrate of potash in the blood, urine, and fæces of persons to whom this salt was exhibited,—the iodide of potassium in the blood, chyle, and urine,—and iodine in the blood of a kid which had been made to respire the vapour of that substance. In other experiments, he found the chloride of barium,—tartar emetic, and nitrate of silver in the blood and fæces. (For the details see Ann. d'Hyg. April 1843, 415.) According to the researches of Dr. Percy, it would appear that alcohol also enters into the blood, and is conveyed to the brain, in which organ, as well as in the liver, he succeeded in detecting it by a common process of distillation. He also found it in the blood, bile, and urine. Still more recently M. Lassaigne has detected ether in the serum of venous blood of animals which had respired ether vapour. By a series of ingenious experiments on the barometric tension of the vapour of the serum before and after the inhalation, compared with the barometric tension of the vapour of ether diluted with water in a known proportion, he estimated that the quantity of ether dissolved in the blood was equal to 0·0008 of its mass. Its effect was to increase the proportion of serum and reduce that of the coagulum (Gas. Méd. 13 Mars, 1847, 209.) It is more easy to determine the presence than the proportion of these substances absorbed into the blood; but we shall see hereafter that the quantity of arsenic and some other poisons may be in certain cases approximately estimated. Almost all those poisons which are susceptible of detection by chemical analysis, have been found by various toxicologists, in the blood, the secretions, or the soft organs of the body of animals to which they have been administered. Cyanide of mercury and chloride of barium have been detected in the blood of the vena portæ and of the splenic vein of the horse. Wöhler found in the urine of dogs and horses iodine, sulphuret of potassium, nitrate of potash, sulphocyanide of potassium, the salts of nickel, the oxalic, tartaric, citric, malic, gallic, succinic and benzoic acids. Orfila has detected arsenious and arsenic acids—the arsenites—the soluble arseniates, tartarised antimony, iodine, potash, barytes and its salts, the mineral acids, sulphuric, nitric, and muriatic—ammonia, muriate of ammonia, and the soluble salts of copper, lead, mercury, gold, and silver. Whether the poison was introduced into the stomach, or applied externally, he equally detected it in the blood (Toxicologie, i. 8.)

Poisons thus absorbed are either deposited in the organs, or slowly eliminated in the secretions, if the individual should survive the effects. These agents appear to fix themselves more in certain organs and secretions than in others. Thus, for example, in cases of arsenical poisoning, the liver, probably from its containing an enormous quantity of blood, and from its proximity to the stomach, is generally more strongly impregnated with arsenic than the other soft organs. The proportion of absorbed arsenic found in it is, according to M. Flandin, nine-tenths of the whole quantity carried into the circulation. When arsenic is not found in the contents of the stomach, and death has taken place within the usual period, it may commonly be detected in the liver. In one case of arsenical poisoning, I was unable to procure any trace of arsenic from four ounces of blood; but procured abundant evidence of the presence of the poison from the examination of the liver. It is important, therefore, in disinterring a dead body for examination, to remove the liver as well as the stomach.

Orfila has found that arsenic most readily passes off, during life, by the urine; while MM. Danger and Flandin think that it escapes by the liver, and pulmonary and cutaneous exhalations (Flandin, i. 568.) They have ascertained that the salts of copper when taken as poisons, are on the other hand, more readily detected in the bronchial secretion than in the urine (Ann. d'Hydg. 1843, 452.) The heart and kidneys were found to contain no copper in animals poisoned by this metal.

The fact that poisons are continually eliminated in the secretions during life, is of considerable importance in reference to a chemical analysis, for, to take arsenic as an example,—if the dose has been small, and the person has survived the effects for a certain period, it is not likely that the poison will be detected in the soft organs of the body. The deceased may have survived long enough for the whole of the poison to be expelled. According to Briand, after ten, twelve, or fifteen days not a particle of arsenic or tartarized antimony will be discovered in the bodies of animals poisoned by either of these substances (Médecine Legale, 437, Ed. 4, 1846.) In a case which occurred to me in November 1846, the individual died eight days after a very *small* dose of arsenic had been given to him. His body had been buried two years. It was disinterred, but on examining most carefully the liver, stomach, pancreas, spleen, and a portion of the small intestines, not a particle of arsenic could be detected. The dose was small, he had survived a long time, and had vomited during the whole period. MM. Danger and Flandin found that in sheep to which a large dose of arsenic (half an ounce) had been given, the poison first appeared in the urine and fæces in about *twenty-two hours;* that it was still discoverable in the urine fifteen days after it had ceased to appear in the fæces, and that it was altogether lost in the excretions, thirty-five days after the ingestion of the poison. When the animal was killed on the thirty-eighth day, not a trace of arsenic could be discovered in its body. (Ann. d'Hyg. 1843, p. 473.) It is difficult to infer from the results obtained by such experiments, the period required for the elimination of the poison from the human system. Even in animals there is a difference. Thus, in young and vigorous dogs arsenic was completely eliminated in from six to ten days, while in sheep the period varied from thirty to thirty-seven days, and the flesh was then safely employed as food. M. Flandin assigns from one to two weeks for its complete disappearance (Des Poisons, i. 738.) In other experiments it entirely disappeared from the body in three days after fifteen grains had been given (i. 737.) The form and dose in which the arsenic is given, as well as the degree to which vomiting and purging have existed during life, must naturally affect the duration of the period at which the poison will be detected in the dead body. In the case of the Queen *v.* Hunter (Liverpool Lent Assizes, 1843) the judge requested one of the medical witnesses to state how long a period was required for the removal of arsenic, by absorption, from the body. There was reason to believe that the deceased had been poisoned: he died in *three* days, but no arsenic was found on an analysis of the contents of the viscera. The tissues were not examined. Some of the witnesses were in-inclined to think that the whole of the arsenic might have been removed from the body in three days, partly by vomiting and purging, and partly by absorption,—the poison being eliminated through the kidneys or the skin. As the tissues were not examined in this case, it was impossible to draw any inference respecting the presence or absence of poison in them. But the experiments of MM. Danger and Flandin show that it is highly improbable that a common dose of this poison should be so rapidly expelled from the body as this hypothesis assumes. The period for the entire disappearance of absorbed arsenic became a question in the case of *Lacoste.* (Flandin, i. 739.) Most of the witnesses assigned the *fourteenth day,* which Flandin regards as the extreme. Orfila assigns from twelve to thirteen days (Toxicol. i. 715,) but the period must in a great measure depend on the time when the poison is entirely expelled from the alimentary canal. The elimination of absorbed arsenic, even when the dose is very small and the poison is administered in solution, *i. e.* in a state most favourable to absorption, appears to be very slow, from a case reported by Dr. Letheby to the Pathological Society of London, in December 1846. A young woman died in thirty-six hours after having swallowed, in two ounces of liquid, a dose of the poison equivalent to two grains and a half of arsenious acid. After death the arsenic (absorbed) was detected in the tissue of the stomach (Med. Gaz. xxxix. p. 116.) As in this case the actual quantity of poison taken was known, as well as the period which the individual survived, it is obvious that, admitting the elimination of the poison not to commence for twelve hours, the quantity which passed off by all the secretions could not have amounted to the *tenth part of a grain* in an hour, or none would have been found in the tissues. From the experiments of M. Bonjean, of Chambery, it would appear that arsenic was detected in the urine of a patient who, *one month before*, had taken in twenty-four days only *three quarters of a grain* of arseniate of soda! (Ann. d'Hyg. 1846, ii. 155.) The proportion thus eliminated may increase after the first day. M. Flandin's experiments on sheep establish this so far as the urine is concerned; but in no instance did they find the quantity of poison thus passed, to exceed the three-hundredths of a grain (·0308 gr.,) even when the dose of arsenic was half an ounce. (Ann d'Hyg. 1843, i. 472.) It appears, therefore, obvious that the quantity of arsenic lost by absorption is inconsiderable; and that it could not account for the disappearance of an ordinary dose of the poison in three days, or even in a week.

Volatile poisons are absorbed and eliminated with very great rapidity. Kramer discovered prussic acid in the blood of an animal which died in *thirty-six seconds*. Dr. Lonsdale found, in experimenting on animals with this poison, that its odour could not be perceived in the blood or in the cavities when life was prolonged beyond *fifteen minutes;* but when death took place within a few minutes, the poison might be detected in the body by the odour for eight or nine days after death. (Ed. Med. and Surg. Journ. vol. li. p. 52.)

This fact shows how much depends on the length of time that an animal survives after taking the poison. It is remarkable, with respect to Ether vapour, that, although most rapidly absorbed, it is but slowly eliminated. In persons who have respired it, the odour has been perceived in the breath for three or four days afterwards.

The facts hitherto stated afford all the evidence that need be desired to prove that a large number of poisons enter the blood. In respect to those which have a local chemical action, this entrance into the circulation by absorption is a mere incident, and by no means necessary to their operation. The nitrate of silver is a corrosive poison, and kills by producing an extensive destruction and disorganization of the viscera. Absorption in this case does not appear to be necessary to its poisonous action, yet it is undoubted that when this substance is exhibited in small doses for medicinal purposes, it is conveyed in some form into the circulation,—a fact established by the peculiar discoloration of the skin of the face and hands, produced by its long-continued employment. It is impossible to say in what form it is transmitted, since, unless the ordinary chemical affinities are suspended by the powers of life, it is not easy to perceive how nitrate of silver could as such be circulated with the albumen or salts of the serum. Some other corrosive poisons, such as potash and the mineral acids, having a purely local action, are no doubt capable of entering into the circulation. With regard to potash, its chemical effects may be soon observed on the urine, although analysis may fail to detect it either in this fluid or in the blood. A case reported by Dr. Letheby, shows that even when sulphuric acid is taken in a concentrated form it is liable to be absorbed and to become eliminated in the urine. A boy aged nine years swallowed an ounce of the acid, and recovered in a few days. For the first four days a large quantity of sulphuric acid was passed with the urine. (Med. Gaz. xxxix. 116.)

The fact, that substances are absorbed which commonly act as poisons independently of this process, might be considered an argument against the necessity for absorption taking place in any case, in order that a poison should produce its usual effects on the body. This question will be presently considered. In the mean time it is quite certain, whatever may be their mode of action, that some poisons find their way into the blood, even when their presence cannot be chemically detected in this fluid. As an additional proof of the extensive diffusion of a poison throughout the body, and its circulation by the blood, may be mentioned the interesting fact, that in the case of a pregnant female poisoned by arsenic in the fourth month of pregnancy, this substance was detected, by MM. Mareska and Lardos, in the body of the fœtus. The poison was also discovered in the uterus and placenta, the latter organ containing a larger proportion than the fœtus, but there was none in the liquor amnii (Gaz. des Hôpitaux, Janvier 1846.) Even the entozoa found in the human body become under these circumstances thoroughly impregnated with the poison. In the case of a female poisoned by arsenic, whose viscera were forwarded to me for examination in July 1845, I found the poison in a worm (*lumbricus teres*) which was discovered dead in the small intestines (Guy's Hosp. Rep. October 1846, p. 462.)

Such, then, are the facts which prove that poisons are absorbed. There are many substances of the absorption of which no proof, chemical or physiological, can be offered; but, judging by analogy, it does not seem unfair to infer, that most if not all poisons can, sooner or later, enter into, and circulate with the blood.

But is this absorption necessary to their fatal action? In some cases, as in the action of the corrosive poisons, the nitrate of silver, the mineral acids and alkalies, it certainly does not appear to be necessary. In other cases, as in the action of arsenic, corrosive sublimate, and alcohol, the question does not admit of so ready an answer.

It might be supposed, that if the blood of a poisoned animal were found to be poisonous, the question would be answered in the affirmative. Vernière has shown by an ingenious experiment, that the venous blood of an animal poisoned by nux vomica is capable of acting as a poison to another; and it is highly probable that if a very large dose of nux vomica could be given to one animal, and, while labouring under its effects, a sufficient quantity of blood could be taken from it and transfused into the body of another, it might be found that this liquid would act as a poison and cause death. There are, however, insuperable obstacles to the performance of such an experiment; because if a large dose of poison be given to the first animal, it may die before a sufficient quantity of blood be transfused from it. If a small quantity of poison be given, or a small quantity of blood be transfused, no fair inference can be drawn from the results; if a large quantity of blood be transfused, this may cause the death of the animal which loses the blood, and yet not be sufficient to produce fatal effects in the other.

Magendie has adopted the view, that all poisons are absorbed, and that the blood is in all cases a solvent of them. According to him, they are transmitted to the brain, and destroy life by directly affecting this organ by contact. Mr. Blake, who limits his definition of the word poison to those substances which appear to destroy life by their effects on the nervous system, has come to the conclusion from experiments on animals, that not only are all poisons absorbed into the blood, but that absorption into the circulation is in every case necessary to their action. This theory, however, as we shall presently see, does not explain all the facts which have been observed and recorded by toxicologists. We cannot exclude from the class of poisons the mineral acids, and yet there is not the least reason to believe that absorption is at all necessary to the action of these bodies.

In the treatment of cases of poisoning, it is necessary to bear in mind, that blood-letting tends to promote the absorption of poisons, and therefore to increase the danger. The administration of emetics which produce nausea, such as tartarised antimony and ipecacuanha, is also considered to be injurious, owing to absorption going on more rapidly while the individual is in this state.

Liquid poisons when swallowed (if we except those which have a local and corrosive action) are more rapidly absorbed than those which are solid. Soluble poisons undergo absorption more rapidly than those which are insoluble, and the larger the quantity of fluid in which the soluble poison is taken, the more speedily it is carried into the circulation. Some solid substances which are but little soluble (arsenious acid) are however very soon absorbed in sufficient proportion to produce well-marked symptoms. Others, which are not very soluble in water, may become dissolved in the acid secretions of the stomach, and be thus absorbed. The carbonate of lead and arsenite of copper are instances of this kind.

SYMPATHY.—When a poison destroys life without apparently entering into the blood, it is said to act through a shock or impulse transmitted from the sentient extremities of the nerves of the part to which it is applied; and this is the theory by which the remote influence of many poisons on vital organs has been explained. It is what is termed an action by sympathy. This view of the action of poisons is founded on the fact that some of these agents produce their effects with too great rapidity to allow of the supposition of absorption being necessary. Thus concentrated hydrocyanic acid in large doses, strychnia, and other alkaloidal poisons, will affect an animal in a few seconds. In exhibiting hydrocyanic acid to three young cats, the symptoms of poisoning came on immediately; and death took place, as nearly as could be ascertained, in from five to ten seconds. Sir B. Brodie and Dr. Christison have also observed this instantaneous action on animals in employing alcohol and muriate of conia. There was no perceptible interval between the contact of the poison and the production of its effects. In some instances, the effect of these powerful agents on the nerves has been actually visible, as in the instance already cited, of the immediate arrest of the peristaltic motion of the intestines by the contact of ticunas. Although when applied to a wound or introduced into the stomach, there is no apparent local change, we cannot doubt, from the rapidity with which the effects ensue, that some impression is produced on the nerves, and transmitted by them to remote organs. The experiments of Addison and Morgan render it probable that the impression is transmitted by the ganglionic, and not by the cerebro-spinal, system of nerves.

An attempt has been made to explain these cases by assuming that the process of absorption is more rapidly carried on than is commonly supposed; and it has been inferred, because hydrocyanic acid and strychnia have been found by physiological experiments to enter the blood of the part to which they are applied in some cases, that this entrance into the circulation is in all instances a condition necessary to their action. With regard to the first point, Müller states that a poison in solution, brought into contact with a wounded surface, may be distributed through the system by absorption in from half a minute to two minutes; but Mr. Blake has inferred from his experiments that a poison may be diffused through the body in so short a period of time as *nine seconds;* and he further asserts that an interval of always more than nine seconds elapses between the introduction of a poison into the capillaries or veins, and the appearance of its first effects. The experiments by which this gentleman endeavoured to show the rapidity with which the blood circulates, do not appear to me to be satisfactory. The mode of experimenting on animals by injecting poisonous liquids into the blood-vessels is liable to give rise to great fallacies, as the animal is placed in an unnatural condition; and no account is taken of the injury which is thus mechanically done by the injection of liquids into the circulation. How little such experiments can be trusted for practical purposes will be obvious from the following fact:—Dr. Christison injected some muriate of conia into the femoral vein of a dog. There was no appreciable *interval* between the moment at which the poison was injected and that in which the animal died: certainly the interval did not exceed *three*, or at most four, seconds (On Poisons, p. 8.) Mr. Blake performed the same experiment; but he found that fifteen seconds elapsed before there were any symptoms, and the animal did not die until *thirty seconds* had elapsed! He repeated this experiment four times, and in no instance did any symptoms manifest themselves in less than fifteen seconds! This was not owing to any want of virulence in the poison; for by no other substance that he used, had death been produced in so short a time (Ed. Med. and Surg. Journ. 53, p. 44.) Here, then, are conflicting results, showing, as it appears to me, that there is something fatal to a correct determination of the rapid action of poisons by these injection-experiments. It is not likely that Dr. Christison should have been mistaken in his estimate of the time; and, therefore, his result, together with similar results of rapid action obtained by others in operating with other poisons, shows that while Mr. Blake's theory, that nine seconds *always* elapse between the introduction of a poison into the capillaries or veins and the appearance of its first effects, may be in accordance with his own experiments, it is inconsistent with the results obtained by others whose observations were made irrespectively of any theory on the subject. Admitting that the absorption of poisons takes place so rapidly as he alleges, it is of course a pure question of fact as to the time at which their effects begin to manifest themselves. In experimenting upon cats with prussic acid, I have seen the effects produced so rapidly, that there was no sensible interval between the application of the poison to the tongue and their production; and death took place in a period of time actually shorter than that which is here stated to be necessary for the appearance of the first symptoms of poisoning. In Freeman's case (see post, poisoning by hydrocyanic acid,) it was stated by the medical witnesses, that a dog *died in three seconds* from the effects of a large dose of hydrocyanic acid. Unless, then, it be rendered probable that a poison may be circulated through the whole of the body in a much shorter period of time than that stated by Mr. Blake, we must admit that these agents do occasionally produce their effects by what is termed sympathy, or by a shock transmitted through the nervous system.

Some poisons appear to act only by absorption, and others independently of that process. Thus, to take two animal poisons, *that* of the rattle-snake produces symptoms instantly, or within a few seconds;—certainly within a period of time so short as not easily to allow of the hypothesis of the poison being diffused by absorption. On the other hand, the poison of hydrophobia by its long incubation appears to act by absorption; for we can hardly imagine, if it acted sympathetically on the nerves by contact, that its operation should be often suspended for so many months. It seems to me that if we are to take the rapid effect of a poison as favourable to the supposition of its action by sympathy, we must take its very slow operation as favourable to the hypothesis that absorption is a state necessary to its action on the system. This would explain why symptoms have not appeared where the bitten part has been early excised. (See HYDROPHOBIA, post.)

[The poison of the rattle-snake is by no means so rapid in its effects as stated by Mr. Taylor, and in most cases there is every evidence of its absorption, as the swelling and pain gradually extend from the wounded part towards the great animal centre, and may be retarded by the application of a ligature between this part and the heart.—G.]

It appears probable that even with the same poison, absorption may be sometimes necessary to its action, and at other times not. Alcohol presents this anomaly. A man has been known to fall senseless instantaneously from a powerful dose of alcohol; in other instances, some minutes have elapsed before the symptoms of poisoning have manifested themselves. Dr. Percy has observed this difference in the effects on animals poisoned by this liquid, but, in the greater number of cases, an interval of a few minutes passed before a total loss of sensibility supervened. Hence he infers, that absorption is generally necessary for the action of alcohol. He has not found that the evacuation of the contents of the stomach by the stomach-pump removed the symptoms—a fact in favour of its acting by absorption. The same has been observed with respect to arsenic; the symptoms have not abated, and persons have died after the poison had been completely removed from the stomach, partly by vomiting, and partly by mechanical means. It is also worthy of remark, that arsenic, when in a state of solution, is very rapidly absorbed. The effects produced by absorbed arsenic are, in some respects, different from those excited by its local contact; and thus we may trace sometimes the exact period of its entrance into the circulation. Faintness, syncope, and general depression, with an indescribable uneasiness, are among the first symptoms caused by absorbed arsenic; and, in a series of cases which I had to examine, these symptoms showed themselves in from five to ten minutes after the poison had been taken in the state of solution (see ARSENIC, post.) Orfila found that when arsenic in solution was injected into the stomachs of dogs, it might be detected in the blood in less than two hours; and in the urine in from six to eight hours (Toxicol. 1, 307, 718; also Galtier, Toxicologie, 1, 318.) It has been supposed that the circumstance of a solid poison being more energetic and speedy in its action, when in a state of solution, was in favour of the view that it acted always by absorption. This, however, is erroneous: it proves nothing either way; for it is obvious that in this finely divided state it is not only better fitted for absorption, but it is, at the same time, better adapted to act on the sentient extremities of the nerves.

Thus, then, I think we may draw these conclusions:—1. That the greater number of poisons are absorbed, and that their remote influence is conveyed through the medium of the blood. 2. That it may also, in certain cases, be conveyed by contact with the sentient extremities of nerves through the nervous system. 3. That some poisons may act in both ways at different times.

CAUSE OF DEATH.—When a poison operates rapidly without entering the blood, death is ascribed to the shock impressed on the general nervous system, from the contact of the poison with the nerves of the living tissues. The nature of the fatal impression thus produced can no more be determined than the nature of thought or sensation. There is no greater difficulty in conceiving that such an impression may be excited by a poison, than that a slight mechanical injury in a remote part of the body may cause an attack of tetanus (Addison and Morgan on Poisonous Agents, p. 64.) The fact that the greater number of poisons enter the blood and act through this fluid, does not bring us any nearer to an explanation of the direct cause of death. One hypothesis assumes that the organ remotely affected is poisoned by the blood which contains the substance dissolved. This doctrine has been lately revived by Liebig in a new form. He considers that the alkaloidal poison—morphia, for example, may be chemically converted into brain by the subtraction of some elements and the addition of others; the quality of the cerebral matter becoming thereby altered, and rendered unfit to support vital energy. (Anl. Chem. 183.) It may be sufficient to say there do not appear to be any grounds for admitting such an extraordinary hypothesis; and that the poison occasionally operates with too great rapidity to allow of the supposition of such a physical change of structure taking place. Anglada supposes that a poison when absorbed, may act directly on the blood by destroying its vitality. Addison and Morgan believe that the poison, when in the blood, acts upon the sentient extremities of the nerves of the lining membrane of the vessels, and that thereby a fatal impression is produced sympathetically on the general nervous system. This theory is supported by those experiments in which death has been caused by the injection of poison into the blood either instantaneously or within a few seconds. Dr. Christison's experiment with muriate of conia appears to admit of explanation only upon this view; for the circulation-theory will not explain the fatal result.

Those who advocate the doctrine of universal absorption in opposition to that of sympathy, suppose that they thereby easily account for the cause of death; but nothing can be more unfounded. Admitting that every poison entered into the blood, and could be chemically detected in this fluid, it would yet remain to be explained *how* it operated when there, to destroy life. At present there is no satisfactory theory to account for the fatal effect. All we know from observation is, that whether the poison be in the midst of the blood in an external wound, or circulating through an artery or vein in the interior of the body, it destroys life. It may be expected that in the progress of microscopical research, the precise effect produced by poisons on the blood will hereafter become a subject of demonstration; but, at present, the *modus operandi* is a perfect mystery. We trace the poison to the circulation, and we observe that death is the result; but neither the chemist nor the microscopist can throw the least light upon the changes produced by the poison in the blood or in the organs necessary to life. Some observers have stated that in arsenical poisoning, the blood is very liquid, as in cases of asphyxia, and that it does not so readily coagulate as in health: but M. Flandin has compared the analysis of healthy blood with that of blood taken from a person poisoned by arsenic, and could perceive no difference in its constitution. (Des Poisons, 1, 560.)

The action of poisons is liable to be modified by habit, idiosyncrasy, and a diseased condition of the body. The influence of disease in increasing or diminishing their action, will be a subject for consideration hereafter.

HABIT, it is well known, diminishes the effect of certain poisons:—thus it is that opium, when frequently taken by a person, loses its effect after a time, and requires to be administered in a much larger dose. Indeed, confirmed opium-eaters have been enabled to take at once, a quantity of the drug which would have infallibly killed them, had they commenced with it in the first instance. Even infants and young children, who are well known to be especially susceptible of the effects of opium, and are liable to be poisoned by very small doses, may, by the influence of habit, be brought to take the drug in very large quantities. This is well illustrated by a statement made by Mr. Grainger, in the Report of the Children's Employment Commission. It appears that the system of drugging children with opium in the Factory districts, commences as soon after birth as possible; and the dose is gradually increased until the child takes from fifteen to twenty drops of laudanum at once! This has the effect of throwing it into a lethargic stupor. Healthy children of the same age would be killed by a dose of five drops. Dr. Christison has remarked that this influence of habit is chiefly confined to poisons derived from the organic kingdom; and I quite agree with him, in thinking that the stories related of arsenic-eaters, and corrosive sublimate-eaters, are not to be credited. There is no proof that any human being has ever accustomed himself, by habit, to take these substances in doses that would prove poisonous to the generality of adults. I have only met with one fact which appears adverse to this opinion. M. Flandin states that he gave to animals doses of arsenious acid in powder, commencing with 1-65th of a grain mixed with their food; and that in nine months, by progressive increase, they bore a dose of upwards of fifteen grains of arsenious acid in powder in twenty-four hours, without their appetite or health becoming affected! (Traité des Poisons, i. 737.) This is contrary to all experience in the medicinal use of arsenic in the human subject; for, as it will be seen hereafter (see ARSENIC,) a very slight increase of a medicinal dose has often been attended with such alarming symptoms, as to render a discontinuance of the medicine absolutely necessary to the safety of the person. The only form in which I have known the question of habit to be raised in medical jurisprudence is this: whether, while the more prominent effects of the poison are thereby diminished, the insidious or latent effects on the constitution are at the same time counteracted. The answer is of some importance in relation to the subject of life-insurance:—for the concealment of the practice of opium-eating by an insured party has already given rise to an action, in which medical evidence on this subject was rendered necessary. As a general principle, we must admit that habit cannot altogether counteract these insidious effects of poisons; but that the practice of taking them is liable to give rise to disease or impair the constitution.

IDIOSYNCRASY differs from habit:—it does not, like this last, diminish the effect of a poison; for it is not commonly found that any particular state of body is a safeguard against the effects of these powerful agents. Some constitutions are observed to be much more affected than others by certain poisons:—thus opium, arsenic, and mercury, are substances of this description, and this difference in their effects is ascribed to idiosyncrasy. Dr. Christison mentions a remarkable instance, in which a gentleman unaccustomed to the use of opium, took nearly an ounce of good laudanum without any effect. (On Poisoning, 33.) This form of idiosyncrasy is very rare. Certain substances generally reputed harmless, and, indeed, used as articles of food, are observed to affect some persons like poisons. This is the case with pork, certain kinds of shellfish, and mushrooms. There may be nothing poisonous in the food itself; but it acts as a poison in particular constitutions:—whether from its being in these cases a poison *per se*, or rendered so during the process of digestion, it is difficult to say. The subject of idiosyncrasy is of great importance in a medico-legal view, when symptoms resembling those of poisoning follow a meal consisting of a particular kind of food. In such a case, without a knowledge of this peculiar condition, we might hastily attribute to poison effects which were really due to another cause. It would appear that in some instances idiosyncrasy may be acquired—*i. e.* a person who, at one period of his life had been in the habit of partaking of a particular kind of food, may find at another period that it will disagree with him. When pork has been disused as an article of diet for many years, it cannot always be resumed by individuals with impunity. When the powers of life become enfeebled by age, the susceptibility of the system to poisons becomes increased; thus aged persons may be killed by comparatively small doses of arsenic and opium. Cases of acquired idiosyncrasy are very rare; it appears to be, if we may so apply the term, a congenital condition.

CHAPTER III.

CLASSIFICATION OF POISONS—SPECIAL CHARACTERS OF IRRITANTS, NARCOTICS, AND NARCOTICO-IRRITANTS—DIFFERENCE BETWEEN CORROSIVE AND IRRITANT POISONS. VARIETIES OF POISONS ARRANGED IN CLASSES.

POISONS were formerly arranged in three classes, according to the kingdom from which they were obtained; and thus we had mineral, animal, and vegetable poisons. The inutility of such a classification must be apparent when it is considered, that we do not, by adopting it, acquire any knowledge of the properties of a poison or of its action on the economy. If applied at all, it should be only in a form subordinate to a physiological classification, so as to allow of the arrangement of poisons in analogous groups. One of the most recent writers on Toxicology, M. Flandin, has endeavoured to revive the old division of poisons into mineral, vegetable, and animal. (Traité des Poisons, i. 225, ed. 1846.) There appears to be no good reason for the reintroduction of this classification, while there are many objections to it. In stating that opium is a narcotic, or that cantharides is an irritant poison, we convey some idea of the mode of action of these substances; but it is not so when we apply to them only the terms *vegetable* and *animal*. We are then left in entire uncertainty as to their mode of operation. All classifications are necessarily more or less arbitrary; but in making our selection, we are bound to prefer that which, while it arranges poisons in a certain order, carries us beyond the mere knowledge of the kingdoms from which they are derived.

Poisons may be divided into three classes, according to their mode of action on the system; namely, IRRITANTS, NARCOTICS, and NARCOTICO-IRRITANTS. This classification is a modification of that originally proposed by Orfila; and is almost universally adopted by toxicologists.

The IRRITANTS are possessed of these common characters. When taken in ordinary doses, they occasion speedily violent vomiting and purging. These symptoms are either accompanied or followed by intense pain in the abdomen. The peculiar effects of the poison are manifested chiefly on the stomach and intestines, which, as their name implies, they irritate and inflame. Many substances belonging to this class of poisons possess corrosive properties, such as the strong mineral acids, caustic alkalies, bromine, corrosive sublimate, and others. These, in the act of swallowing, are commonly accompanied by an acrid or burning taste, extending from the mouth down the œsophagus to the stomach. Some irritants do not possess any corrosive action,—of which we have examples in arsenic, the poisonous salts of barytes, carbonate of lead, cantharides, &c., and these are often called pure irritants. They exert no chemical action on the tissues with which they come in contact; they simply irritate and inflame them.

There is this difference between CORROSIVE and IRRITANT poisons. Under the action of corrosive poisons, the symptoms are commonly manifested immediately, because mere contact produces disorganization of a part, usually indicated by some well-marked symptoms. In the action of the purely irritant poisons, the symptoms are generally more slowly manifested, rarely showing themselves until at least half an hour has elapsed from the time of swallowing the substance. Of course, there are exceptions to this remark; for sometimes irritants act speedily, though seldom with the rapidity of corrosive poisons. It is important, in a practical view, to distinguish whether in an unknown case, the poison which a person, requiring immediate treatment, may have swallowed, be irritant or corrosive. This may be commonly determined by the answer to the question, as to the time at which the symptoms appeared after the suspected poison was taken. In this way we may often easily distinguish between a case of poisoning from arsenic and one from corrosive sublimate. There is also another point which may be noticed. As the corrosion is due to a decided chemical action, so an examination of the mouth and fauces may enable us to determine the nature of the poison swallowed.

It has been already stated that there are many irritant poisons which have no corrosive properties, and therefore never act as corrosives; but it must be remembered that every corrosive may act as an irritant. Thus the action of corrosive sublimate is that of an irritant poison, as while it destroys some parts of the coats of the stomach and intestines, it irritates and inflames others. So again most corrosive poisons may lose their corrosive properties by dilution with water, and then they act simply as irritants. This is the case with the mineral acids, and bromine. In some instances, it is not easy to say whether an irritant poison possesses corrosive properties or not. Thus oxalic acid acts immediately, and blanches and softens the mucous membrane of the mouth and fauces, but I have never met with any decided marks of what could be called chemical corrosion produced by it in the stomach or viscera.

Irritant poisons, for the most part, belong to the mineral kingdom; and they may be divided into the non-metallic and metallic irritants. There are a few derived from the animal and vegetable kingdom; but these are not very often employed criminally. Some of the gases likewise belong to the class of irritant poisons.

Narcotic poisons have their operation confined to the brain and spinal marrow. Either immediately or some time after the poison has been swallowed, the patient suffers from cephalalgia, vertigo, paralysis, coma, and in some instances tetanus. They have no acrid burning taste like the corrosive irritants; and they very rarely give rise to vomiting or diarrhœa. When these symptoms follow the ingestion of the poison into the stomach, the effect may be ascribed either to the quantity in which the poison has been taken, and the mechanical distention of the stomach thereby produced, or to the poison being combined with some irritating substance, such as alcohol. The pure narcotics are not found to irritate or inflame the viscera.

Notwithstanding the well-defined boundary thus apparently existing between these two classes of poisons, it must not be supposed that each class of bodies will always act in the manner indicated. Some irritants have been observed to affect the brain or the spinal marrow remotely. This is the case with oxalic acid and arsenic. Both of these common poisons have in some instances given rise to symptoms closely resembling those of narcotic poisoning; namely coma, paralysis, and tetanic convulsions. I have met with one case of poisoning by arsenic in which there was paralysis of the extremities with an entire absence of purging during the eight days which the deceased survived. Thus, then, we must not allow ourselves to be deceived by the idea that the symptoms are always clearly indicative of the kind of poison taken. The narcotic poisons are few in number, and belong to the vegetable kingdom. Some of the poisonous gases possess a narcotic action.

Narcotico-Irritants.—Poisons belonging to this class have, as the name implies, a compound action. They are chiefly derived from the vegetable kingdom. At variable periods after being swallowed, they give rise to vomiting and diarrhœa like irritants; and sooner or later produce stupor, coma, paralysis and convulsions, owing to their effect on the brain and spinal marrow. They possess the property, like irritants, of irritating and inflaming the alimentary canal. As familiar examples we may point to nux vomica, monkshood, and poisonous mushrooms. This class of poisons is very numerous, embracing a large variety of well-known vegetable substances; but they rarely form a subject of difficulty to a medical practitioner. The fact of the symptoms occurring after a meal at which some suspicious vegetables may have been eaten, coupled with the nature of the symptoms themselves, will commonly indicate the class to which the poison belongs. Some narcotico-irritants have a hot acrid taste, such as the aconite or monkshood.

I here subjoin tables of the more important poisons, with the properties of which it is necessary for a medical jurist to be acquainted. Poisons are divided into three classes—

IRRITANTS **NARCOTICS** **NARCOTICO-IRRITANTS.**

IRRITANT poisons may be divided into

MINERAL VEGETABLE ANIMAL

and MINERAL IRRITANT POISONS may be subdivided into Non-metallic and Metallic.

Class I.

IRRITANT POISONS.

1. NON-METALLIC IRRITANTS.

Acids.—Sulphuric acid. Aromatic sulphuric acid. Sulphate of indigo. Nitric acid. Muriatic acid. Phosphoric acid. Nitro-muriatic acid. Nitro-sulphuric acid. Oxalic acid. Tartaric acid. Citric acid. Acetic acid. Vinegar.

Alkalies.—Potash and its carbonates. Soda and its carbonates. Ammonia and its carbonates. Baryta. Strontia. Lime.

Alkaline Salts.—Binoxalate of potash. Bitartrate of potash. Bromide of potassium. Iodide of potassium. Sulphurets of potassium and sodium. Persulphuret of calcium. Alkaline hypochlorites (chlorides of potash, soda, and lime.) Muriate of ammonia. Nitrate of potash. Sulphate of potash. Sulphate of alumina and potash (alum.) Chloride of sodium (common salt.)

Metalloidal Poisons.—Phosphorus. Chlorine. Bromine. Iodine. Sulphur.

2. METALLIC IRRITANTS.

ARSENIC. Arsenious acid. Arsenite of potash (Fowler's solution.) Metallic arsenic. Fly powder. Fly water. Arsenic acid. Arseniates. Sulphurets of arsenic (orpiment, realgar.) Iodide and bromide of arsenic. Arsenuretted hydrogen gas. MERCURY. Corrosive sublimate. Calomel. Ammonio-chloride of mercury (white precipitate.) Black oxide of mercury. Mercurial ointment. Mercury with chalk (grey powder.) Red oxide of mercury. Red precipitate. Iodide of mercury. Cinnabar. Vermilion. Bicyanide of mercury. Turbith mineral. Nitrates of mercury. Acetate of mercury. LEAD. Acetate (sugar of lead.) Goulard's extract. Nitrate of lead. Chloride of lead. Oxychloride (Turner's yellow.) Carbonate of lead (white lead.) Sulphate. Chromate. Iodide. Oxides. COPPER. Alloys of copper. Sulphate (blue vitriol.) Subacetate (verdigris.) Oxychloride. Phosphate. Sulphuret. Ammonio-sulphate. Nitrate. Carbonate. Oxides. Verditer. ANTIMONY. Tartarised antimony. Chloride. Sulphurets. Oxides. ZINC. Sulphate (white vitriol.) Acetate. Carbonate (calamine.) TIN. Chlorides. Dyer's spirit. SILVER. Nitrate (lunar caustic.) GOLD. Terchloride. IRON. Sulphate (copperas or green vitriol). Muriate. BISMUTH. Subnitrate. CHROME. Bichromate of potash.

3. VEGETABLE IRRITANTS.

Aloes. Anemone. Arum. Bryony. Castor-oil seeds. Cayenne pepper. Celandine. Colocynth. Creasote. Croton seeds and oil. Daffodil. Elaterium. Elder. Euphorbium. Gamboge. Hyssop. Jatropha (Curcas.) Jalap. Manchineel. Mezereon. Mustard. Ranunculus. Savin. Scammony. Stavesacre. Caltha palustris. Clematis vitalba. Cochlearia armoracia. Cyclamen Europæum. Fritillaria imperialis. Lobelia syphilitica. Plumbago Europæa. Rhus toxicodendron. Rhus radicans. Rhododendron chrysanthum. Sedum acre. Oil of tar. Oil of turpentine. Pyroxylic spirit. Dippel's oil. Fusel oil. Decayed vegetable matter. Decayed carrots. Potatoes. Mould. Mouldy bread. Flour. Ergot of rye.

4. ANIMAL IRRITANTS AND ANIMAL POISONS.

Cantharides. Poisonous food. Fish poison. The Muscle. Whelk. Oyster. Periwinkle. Lobster. Crab. Pickled salmon. Herring. Halibut. Mackerel. Tunny. Cheese. Sausages. Train oil. Flesh of animals poisoned from disease or decay. Bacon. Diseased and putrified animal matter. Farcinoma (Glanders.) Poison of the dead body. Poison of rabies (Hydrophobia.) Poison of venomous serpents. The viper. Rattle-snake. Cobra di Capello. Venomous insects. Scorpion. Scolopendra. Spider. Tarantula. Argos Persicus. Bee. Wasp. Hornet.

Class. II.

NARCOTIC POISONS.

Opium. Morphia and its salts. Hydrocyanic acid. Bitter almond. Cherry laurel. Jatropha manihot. Cyanide of potassium. Hyoscyamus. Lactuca. Solanum. Camphor. Alcohol. Ether.

Class III.

NARCOTICO-IRRITANT POISONS.

Carbazotic acid. Cevadilla. Cocculus Indicus. Coriaria myrtifolia. Darnel grass. Foxglove. Hellebore. Hemlock. Ipecacuanha. Laburnum. Lobelia inflata. Meadow-saffron. Monkshood. Mushrooms. Nightshade. Nux vomica (Strychnia.) Rue. Squill. Thornapple. Ticunas. Tobacco. Upas. Woorara. Yew.

POISONOUS GASES.

Ammonia. Carbonic acid. Carbonic oxide. Carburetted hydrogen (coal-gas.) Chlorine. Cyanogen. Hydrogen. Muriatic acid. Nitrogen. Protoxide of nitrogen. Deutoxide of nitrogen. Sulphuretted hydrogen. Sulphurous acid.

CHAPTER IV.

EVIDENCE OF POISONING IN THE LIVING SUBJECT—SYMPTOMS OCCUR SUDDENLY—CAUSES OF RETARDATION OF SYMPTOMS—ACTION OF POISONS AGGRAVATED BY DISEASE—SYMPTOMS CONNECTED WITH FOOD OR MEDICINE—SUDDEN DEATH FROM NATURAL CAUSES MISTAKEN FOR POISONING—SEVERAL PERSONS ATTACKED SIMULTANEOUSLY—EVIDENCE FROM THE DETECTION OF POISON IN FOOD.

We shall next proceed to consider the evidence of poisoning in the living subject. To the practitioner the diagnosis of a case of poisoning is of very great importance, as by mistaking the symptoms produced by a poison for those arising from natural disease, he may omit to employ those remedial measures which have been found efficacious in counteracting its effects, and thus lead to the certain death of the patient. To a medical jurist a correct knowledge of the symptoms furnishes the chief evidence of poisoning, in those cases in which persons are charged with the criminal administration of poison with intent to murder, but from the effects of which the patient ultimately recovers. The symptoms produced during life, constitute also an important part of evidence, in those instances in which the poison proves fatal. At present, however, we will suppose the case to have been, that poison has been taken and the patient survives.

Most toxicological writers have laid down certain characters whereby it is said symptoms of poisoning may be distinguished from those of disease.

1. IN POISONING, THE SYMPTOMS APPEAR SUDDENLY, WHILE THE INDIVIDUAL IS IN HEALTH.—It is the common character of most poisons, when taken in the large dose in which they are usually administered with criminal intent, to produce serious symptoms either immediately or within a very short period after they have been swallowed. Their operation, under such circumstaces, cannot be suspended, and then manifest itself after an indefinite interval; although this was formerly a matter of universal belief, and gave rise to many absurd accounts of what was termed *slow* poisoning. In modern times, the negroes of Martinique have been said to possess this art, but the late interesting researches of Dr. Rufz show that this is an erroneous statement. (Annales d'Hygiène, 1844, i. 392; also ii. 170.) It is very true, that these powerful agents, given at intervals in small doses, do not cause those striking symptoms upon which a practitioner commonly relies as evidence of poisoning. They may then produce disorder, but of so slight a nature, as scarcely to excite suspicion. In fact, under these circumstances, the symptoms often so closely resemble those of disease, that an inexperienced practitioner may be easily mistaken respecting their origin, especially where no circumstances exist to create the least suspicion of criminality on the part of relatives and others around the patient. Arsenic given in small doses, at long intervals, has thus occasioned symptoms resembling those which depend on disease of the stomach. After several attacks and recoveries suspicion may be completely disarmed. In November 1846, a case of this kind was referred to me for investigation, in which it was alleged that a farmer in one of the midland counties had been poisoned two years before by his housekeeper, who was a respectable person, and most attentive to him as a nurse during his illness. He had been attacked with vomiting and other signs of gastric disorder, about three months before his death, but recovered under medical treatment. About eight days before death the symptoms recurred with greater violence than ever, and he sank under them. They were referred to ulceration of the stomach, so closely did they resemble those of disease. As there was no suspicion of poison, the body was not examined; and nothing would have been known respecting the real cause of death, but for a statement made two years afterwards, by the housekeeper, that she had on two occasions administered to her master small doses of arsenic, and the last, probably from its being larger than the first, had occasioned death. Again, there are what are called *accumulative* poisons,—substances which, in small divided doses, given at long interval, produce scarcely any perceptible effect on the system; but which appear to accumulate in the body, and at some unexpected time their power is manifested with sudden and violent energy. To these forms of poisoning, which it is extremely rare to meet with on criminal charges, the characters about to be described are not applicable.

When poison is criminally administered, it is almost always in such doses as to cause the symptoms to appear *suddenly*, and to run their course with great rapidity. The symptoms of poisoning by prussic acid, oxalic acid, or strychnia, appear immediately, or within a very few minutes after the poison has been swallowed. In one case, however, where the dose of prussic acid was small, and insufficient to produce death, the poison was supposed by the patient not to have begun to act until after the lapse of fifteen minutes. (Ed. Med. and Surg. Journ. lix. 72.) The symptoms caused by arsenic and other irritants, and, indeed, by all poisons generally, are commonly manifested in from half an hour to an hour. It is rare that the appearance of the symptoms is protracted for two hours, except under certain peculiar states of the system. It is said, that some narcotico-irritant poisons, such as the poisonous mushrooms, may remain in the stomach twelve or twenty-four hours without giving rise to symptoms; and this is also affirmed to be the case with some animal irritants, such as decayed meat; but with regard to the first point, it has been shown by Dr. Peddie, that mushrooms may produce symptoms in half an hour; and a case has fallen under my own observation, where the symptoms from noxious food come on within as short a time after the meal, as is commonly observed in irritant poisoning by mineral substances. In cases of poisoning by phosphorus, the symptoms do not commonly begin until after the lapse of many hours.

Influence of sleep.—The symptoms produced by some of the most common poisons, are apt to be retarded under certain conditions of the system. When an irritant poison is taken on a full stomach, the symptoms do not commonly appear so speedily as when the stomach is empty. So again, it is stated by Dr. Christison, from cases which have fallen under his notice, that *sleep* retards the action of arsenic, and the same may hold with other irritants. Thus, if the patient should happen to fall asleep soon after swallowing the poison, it may not produce the usual symptoms until four or five hours afterwards, or the occurrence of these may be even longer protracted. This is supposed to be owing to the general state of insensibility of the body, and the depressed condition of the nervous system during sleep.

Influence of intoxication.—This state has been considered to retard the operation of opium. Observations of this kind must, of course, be accidental; and there is scarcely a sufficient number of cases reported of narcotic poisoning under these circumstances, to justify a decided opinion on the point. It was observed of a person who had swallowed a strong dose of opium, while partially intoxicated, that the symptoms were some hours before they were manifested. Perhaps, strictly speaking, the symptoms in these cases are masked.

Influence of disease.—A *diseased* state of the body may render a person comparatively unsusceptible of the action of some poisons, while in other instances it may increase their action, and render them fatal in small doses. In dysentery and tetanus, a person will take, without being materially affected, a quantity of opium sufficient to kill an adult in average health. Mania, cholera, hysteria, and delirium tremens, are also diseases in which large doses of opium may be borne with comparative impunity. In a case of hemiplegia, a woman, æt. 29, took for six days, three grains of strychnia daily without injurious consequences—the dose having been gradually raised (Gaz. Med. Mai 1845;) while one grain of strychnia is commonly regarded as a fatal dose to a healthy person. In a case of tetanus, Dupuytren gave as much as two ounces of opium at a dose (60 grammes,) without serious consequences. (Flandin, Traité des Poisons, i. 231.) It has also been remarked, that persons affected with tetanus are not easily salivated by mercury. (Colles's Lectures, i. 77.) The effect of certain diseases of the nervous system as well as of habit, either in retarding the appearance of symptoms, or in blunting the operation of a poison, it is not difficult to appreciate; and they are cases which can present no practical difficulty to a medical jurist. On the other hand, in certain diseased states of the system, there is an increased susceptibility to the action of poison. Thus, in those persons who have a tendency to apoplexy, a small dose of opium may act more quickly and prove fatal. In a person labouring under inflammation of the stomach or bowels, there would be an increased susceptibility to the action of arsenic or other irritants. One of the most remarkable instances of the influence of disease in increasing the operation of poison, is perhaps seen in cases of diseased kidney (granular degeneration,) in which very small doses of mercury have been observed to produce severe salivation, leading to exhaustion and death. A knowledge of this fact is of importance in referenee to charges of malapraxis, when death has arisen from ordinary doses of calomel administered to persons labouring under this disease.

A medico-legal case in which this question arose, will be found fully reported in Guy's Hospital Reports, Vol. iv. Oct. 1846, p. 443. As a general principle it may be affirmed, that whenever the body is much debilitated by disease, poisons acquire greater virulence of action. A most absurd degree of importance appears to have been attached to this modifying condition, in *Tawell's* case (Bucks Lent Assizes, 1845.) It was, as I am informed, alleged that epilepsy would increase the effects of prussic acid, so as to render this poison more rapidly fatal in a smaller dose. In the accident which occurred to the seven Parisian epileptics, on which so much stress was laid in the defence, one patient died in about twenty minutes, and another lived three-quarters of an hour although all took the *same dose*, which it was afterwards contended was unusually large. These facts connected with the influence of disease are obviously of some importance in relation to those cases where the party who has taken the poison is already in a diseased or exhausted state. Thus, then, there are but few exceptions to the rule laid down, that the symptoms of poisoning are liable to appear suddenly; and that in most cases they are commonly manifested within an hour.

Symptoms appear during a state of health.—Symptoms of poisoning often manifest themselves in a person while in a state of *perfect health*, without any apparent cause. This rule, of course, is open to numerous exceptions, because the person on whose life the attempt is made, may be actually labouring under disease; and under these circumstances, the symptoms of poisoning are so obscure as often to disarm all suspicion. In the case of *Mrs. Smith*, who was poisoned by orpiment in 1835, it was the fact of the deceased having laboured under general illness for some time before death, that prevented any suspicion from being fixed on the prisoner Burdock, who attended her as a nurse. When poison is exhibited in medicine, a practitioner is very liable to be deceived, especially if the disease under which the party is labouring be of an acute nature, and attended by symptoms of disorder in the alimentary canal. Several cases of poisoning have occurred within the last few years, where arsenic was criminally substituted for medicine, and given to the parties while labouring under a disorder of the bowels. Thus, then, it may be said with respect to this character of poisoning, that when in a previously healthy person, violent vomiting and purging occur suddenly, and without any assignable cause, such as disease or indiscretion in diet, to account for them, there is strong reason to suspect that irritant poison has been taken. When the party is already labouring under disease, we must be especially watchful on the occurrence of any sudden change in the character or violence of the symptoms, unless such change can be easily accounted for on common or well-known medical principles. In most cases of criminal poisoning, we meet with alarming symptoms without any obvious or sufficient natural cause to explain them. The practitioner is of course aware that there are certain diseases which are liable to occur suddenly in healthy people, the exact cause of which may not at first sight be apparent; therefore this criterion is only one out of many on which a medical opinion should be founded. Some have said that the symptoms of poisoning are characterized by a *regularity* of increase, or by becoming more and more aggravated as the case advances; but this is a weak criterion. In the operation of most of the active irritants, there are often remissions, and occasionally intermissions of pain, so as to give rise to a false hope of recovery. It must not therefore be inferred that a recurrence of the symptoms of irritation is necessarily indicative of the administration of a fresh dose of poison. The character of the symptoms is in other cases suddenly changed; vomiting may cease, and may be succeeded by coma. While, then, on the one hand, such a case might, by our trusting too much to this criterion, be regarded as one rather of disease than of poisoning; there are, on the other hand, certain diseases which are very rapid and violent in their progress, and the symptoms of these might, for the same reason, be mistaken for those of poisoning.

2. IN POISONING, THE SYMPTOMS APPEAR SOON AFTER A MEAL, OR SOON AFTER SOME KIND OF FOOD OR MEDICINE HAS BEEN TAKEN.—This is by far the most important character of poisoning in the living body. It has been already observed, that most poisons begin to operate within about an hour after they have been swallowed; and although there are some few exceptions to this remark, yet they occur under circumstances easily to be appreciated by a practitioner. Thus, then, it follows, that, supposing the symptoms under which a person is labouring, to depend on poison, the substance has most probably been swallowed either in food or medicine, from half an hour to an hour previously. It must be observed, however, that cases of poisoning may occur without the poison being introduced by the mouth. Oil of vitriol has been thrown up the rectum in the form of enema, and caused death: the external application of arsenic, corrosive sublimate, and cantharides has destroyed life. In one case arsenic was introduced into the vagina of a female, and she died in five days under all the symptoms of arsenical poisoning. (Schneider, Ann. der Ges. Staatsarzneikunde, i. 229.) Such cases are rare, but nevertheless the certainty that they have occurred where their occurrence could hardly have been anticipated, shows that in a suspicious case, a practitioner should not deny the fact of poisoning, merely because it is proved that the patient could not have taken the poison in the usual way—by deglutition.

Let us suppose, however, the circumstances to have been such that these secret means of destruction could not have been resorted to, and that the poison is one of those most commonly selected by a murderer, such as arsenic, oxalic acid, or corrosive sublimate; then we may expect that this character of poisoning will be made evident to us, and that something must have been swallowed by the patient shortly before these alarming symptoms appeared. By observations attentively made, it may be in our power to connect the appearance of the symptoms with a particular article of food, and thus indirectly lead to the detection of the criminal. Supposing that many hours have passed since food or medicine was taken by the patient, without any effect ensuing,—it becomes very probable that the symptoms are due to some other cause, and not to poison. The *time of the occurrence of the symptoms* in relation to a particular meal, is then a fact of especial importance in forming an opinion when poisoning is suspected, as the following cases will show.

The *Crown Prince of Sweden* was considered by many to have been killed by poison. The prince, it appears, was reviewing some troops, when he was observed to fall suddenly from his horse, and he died half an hour afterwards. His physician, Dr. Rossi, was accused of having administered poison to him, and was obliged for his own security to quit the country. It is obvious, however, from an examination of the particulars of the case, that had this sudden attack been due to poison, it could only have been from one of the most active narcotics, given to him but a short time before he fell from his horse. But it was ascertained that the prince had taken neither solid nor liquid of any kind for at least *four hours* previously to his death. The allegation of poisoning was thus disproved, for no poison operating with symptoms like those under which the prince had died could have had its effects suspended for four hours. The cause of death was apoplexy.

The following case, mentioned by Dr. Christison, is also of some interest. A middle-aged man retired to bed one afternoon about two o'clock, much fatigued. In about a quarter of an hour, he was found by one of his workmen, gasping, rolling his eyes, and quite insensible; he died a few minutes afterwards. As he had lived unhappily with his wife, it was suspected that she had poisoned him; and the body was therefore examined. The only morbid appearance met with was a considerable tuberculation of the septum and parietes of the ventricles of the heart. This was probably the cause of death: had there been any doubt on the point, the circumstances attending the man's death would at least have shown that it was impossible he could have died from poison. The deceased had taken nothing since his breakfast at nine o'clock. No poison, but one of the most active narcotics in a large dose, given but a short time before the attack, could have caused death in a quarter of an hour. These active poisons cannot have their effects suspended for *five hours*. It was, therefore, clear, without resorting to a chemical analysis of the contents of the stomach, that the deceased could not have died from poison.

In February 1845, the following case was submitted to me by my friend Mr. J. G. French. A child between two and three years of age, in tolerable health, was one afternoon suddenly seized with stupor, convulsions, and insensibility, and died in twenty-three hours. After death the brain was found highly congested. All suspicion of narcotic poisoning was done away with by the fact that the child had taken nothing since its dinner at two o'clock, and the symptoms suddenly appeared at half-past five, *i. e.* three hours and a half afterwards. (Med. Gaz. xxxvi. 32.)

The following case of sudden death under symptoms resembling those caused by irritant poison, is also of interest. At three P. M., on the 19th of January, a child, aged eleven, was found by a medical practitioner who was called to see her, in a state of collapse; pulse at the wrist scarcely to be felt; extremities cold: countenance livid. She was quite sensible, and complained of pain over the abdomen, which was full, and evidently contained fluid. The pain was not increased by pressure. Stimulants were administered. Three hours after the visit she was dead. The parents gave the following history of the case. In August last she had an erysipelatous affection of one leg, which disappeared after the use of some aperient medicines. Soon after, she began to complain of pain over the abdomen, with loss of appetite. The pain was never severe, neither was her general health so much disturbed as to confine her to the house, or to induce her friends to apply for medical advice. After five or six weeks, her body began to enlarge; she had thirst, and became indifferent to motion, and was soon fatigued by any bodily exertion. The abdominal pains were now trifling, recurring only at intervals. She continued in this state till December, when she left home. During her absence her appetite improved, but her body continued large, and her strength declined. She returned to her friends on the 17th of January. On the 18th she ate a hearty dinner with the family, and seemed better than usual. At five P. M., about four hours after dinner, she was seized with violent pain over the abdomen, and vomiting. Collapse ensued and she died in twenty-five hours. The stomach and intestines were found healthy, and contained only partially digested food. (Prov. Journal, Feb. 19, 1845, p. 114.)

If in either of these four cases the symptoms had supervened shortly after food had been taken, it is easy to understand that they might have been referred to poison. By bearing in mind these facts, regarding the period at which the more common poisons begin to produce their effects, it may often be in our power to determine summarily, without a chemical analysis, whether the case be one of poisoning or not. In several instances which have been brought to Guy's Hospital, where narcotic poison was suspected to have been the cause of comatose symptoms and rapid death, there was no difficulty in deciding against the suspicion of poisoning, merely from observing the circumstances under which the attack took place. Facts of this kind may sometimes serve to establish the innocence of an accused party, and at others to point out the real criminal.

A woman aged sixty-five, accused her husband, an old man of seventy, of having attempted to poison her. The woman was passionate, ill-tempered, eccentric in her habits, and subject to occasional attacks of hysteria. She handed to the authorities a vessel containing arsenic in coarse powder; and some food which she stated had been prepared for her by the prisoner. On analysis, the food was found to contain a large quantity of arsenic. The husband was immediately committed to prison. The wife left her bed, and was apparently quite well; and so she remained for eight days afterwards, no symptoms of poisoning having manifested themselves about her. She was then seized with a fit of mania, and was guilty of many extravagant acts. She died the following day, *i. e.* nine days after she had accused her husband of having administered arsenic to her in her food. On a post-mortem examination, it was evident she had died from the effects of arsenic. This poison was found in large quantity in the alimentary canal; and there were the usual morbid changes in the stomach and intestines. The husband denied that he had administered poison to the deceased. This denial, however, would have availed him but little, had it not been for the careful medico-legal investigation of the whole case, made by the medical witnesses. As the husband had been confined in prison *eight days* before the death of his wife, he could not have committed the crime imputed to him, unless he had administered the arsenic previous to his imprisonment. His guilt, therefore, rested upon the medical question, whether a large quantity of arsenic could be taken by a person and remain dormant in the system, without producing any of its usual effects for the long period of eight days? The witnesses very properly answered the question in the negative, and the husband was immediately discharged. (Annales d'Hygiène, 1836, ii. 391.) While the prisoner was with his wife, she did not suffer from the symptoms of poisoning, nor was there any proof that he had administered poison. When, however, he was so situated that he could not possibly have been accessory to its administration, she died from its effects. It was fortunate for the accused that he was thrown into prison, and that the case fell into the hands of persons versed in the subject of legal medicine.

The following singular case is related by Alison:—

Jean Aitkin, or *Humphreys*, was tried at the Aberdeen September Circuit, 1830, for the murder of her husband, by pouring sulphuric acid down his throat as he lay asleep in bed. The parties frequently quarrelled, and were both addicted to habits of intoxication. On the night in question, some friends had passed the evening with them drinking. They went away about twelve o'clock at night, and soon after this, the deceased was seen asleep in bed. The only persons in the house at this time were the prisoner and a servant-maid, and the street door was locked so that no other person could have access. The prisoner left the servant's room on her stocking-soles, a thing unusual for her, and when she returned in about twenty minutes, she told the servant that her husband was roaring mad with drink. The girl, upon going to him, found him lying upon his back, declaring he was all roasting. The prisoner at first showed an unwillingness to send for a medical man, but at length did so. When the deceased left the guests at twelve, there were only two glasses on the table in the room; but when the neighbours came in after the alarm, there were *three*, and the third was proved to have come from a room above stairs, of which the prisoner had the key. This glass contained, it was supposed, sulphuric acid. In the room where the deceased was lying there was a vial which had contained sulphuric acid, but it was then nearly empty. The deceased lived two days, but never could give any further account of the matter than that he went to sleep quite well, and awoke "all roasting," and had suffered the utmost agony ever since. He evidently died from the effects of sulphuric acid, large quantities of which were detected on his shirt, on the blanket and bedcover, and a little on the prisoner's bedgown and handkerchief; but not a trace of the poison could be discovered in the stomach or intestines of the deceased. (Alison, Criminal Law of Scotland, p. 75.) For an able medico-legal analysis of this case by Dr. Christison, see Medical Gazette, vol. viii. p. 77.

The defence pleaded that the deceased had voluntarily taken the poison and committed suicide; but the only time at which he could by any possibility have taken it, was when he was drinking with his friends; for immediately after they left, he went to bed, and was seen asleep; and, according to his own account, he awoke suddenly with the pain, and other symptoms produced by this poison. It was impossible that he could have swallowed the acid while drinking with his friends; for the symptoms of the corrosives comes on *suddenly*, and cannot be suspended; therefore the poison must have been poured down his throat while he was sleeping, and as the house was at that time fastened up, this act could only have been perpetrated by the prisoner or the maid-servant. The circumstances above mentioned clearly showed that the prisoner was the guilty party. It will be observed that all suspicion of suicide, as well as of murder on the part of the persons with whom the deceased had been drinking, was entirely removed, by attention being paid to this well-marked character of the corrosive poisons.

When symptoms resembling those of poisoning speedily follow the ingestion of food or medicine, there is, however, always great room for suspicion; but caution should be observed in drawing inferences, since the most extraordinary coincidences sometimes present themselves. In the celebrated case of *Sir Theodosius Boughton*, who was poisoned by his brother-in-law, Donellan, in 1781, the fact of alarming symptoms coming on in *two minutes* after the deceased had swallowed what was supposed to be a simple medical draught, became the most important evidence against the prisoner. There is no doubt that laurel-water had been substituted for the medicine by the prisoner. I may here remark, that the practice of substituting poisonous mixtures for medical draughts or powders, is by no means unusual, although it might be supposed to indicate a degree of refinement and knowledge not commonly to be found among the lower classes of criminals. Medical practitioners are thus apt to be imposed upon, and the following case, related by one of our present judges, will serve as a caution. An apothecary prepared a draught, into which another person put poison, intending thereby to destroy the life of the patient for whom the medicine was prescribed. The patient, not liking the taste of the draught, and thinking that there was something suspicious about it, sent it back to the apothecary, who, knowing the ingredients of which he had composed it, and wishing to prove to his patient that he had done nothing wrong, drank it himself, and died. In this case, he was the unconscious agent of his own death; and although the draught was intended for another, the party who poisoned it was held guilty of murder. This case contains a serious warning to medical witnesses. It is not very unusual on trials for poisoning, when the poison is conveyed through medicine, to find a medical witness offering to swallow his own draughts in a court of law, in order to furnish to the court and jury a convincing practical illustration of the innocence of the medicine! I need hardly observe that an exhibition of this kind is never required of a medical witness. The court will receive his deposition, without compelling him to swallow his own medicine, even supposing it not to have been secretly poisoned. If any doubt be raised of the innocent properties of the draught, a chemical analysis of its contents will be far more satisfactory, and attended with no kind of risk to the practitioner.

In a case which I had to examine in February 1847, a mixture of soap liniment and opium had been substituted for the tincture of sesquichloride of iron, prescribed for a female many months before. Some of the liniment had been given to a young infant, and, as it was alleged, had led to its death. The mother was tried and acquitted upon the charge of murder at the Essex Lent Assizes, 1847, the cause of death not being clearly traced to the action of the opiate.

(*Queen* v. *Gray* and *Bright*.) The defence was, that a mistake had been made in dispensing the medicine; but the fact that the phial had originally contained an iron mixture, was proved by the discovery of iron in some brown stains upon the label, owing to a portion of the liquid having been accidentally spilled over it. The cork was also blackened from the gallic acid and tannin contained in it.

On the other hand, the occurrence of symptoms resembling those produced by poison, soon after food or medicine has been taken, may be a pure coincidence. In such a case, poison is always suspected by the vulgar; and it will be the duty of a medical jurist to guard against the encouragement of such a suspicion, until he has strong grounds to believe it to be well founded. No public retractation or apology can ever make amends for the injury which may in this way be inflicted on the reputation of another; for those who hear the accusation, may never hear the defence. In all such cases, a practitioner may entertain a suspicion, but he should always avoid *expressing* it or giving it publicity. When death is not a consequence, it is difficult to clear up such cases, except by the aid of a chemical analysis; but this, as we know, is not always applicable. If death ensue, the real cause is usually apparent, and a suspicion of poisoning is thus often removed by a post-mortem examination.

The fatal symptoms produced by perforation of the stomach, which in some respects resemble those of arsenical poisoning, almost always attack an individual soon after a meal. When they occur some hours after, there is less likelihood of confounding them with arsenic. A few years ago, Mr. Hilton, of Guy's Hospital, and myself, had to examine a case of this description. The diagnosis was in a great measure aided by the fact, that the violent symptoms did not appear until about three hours after a meal. An instance occurred within my knowledge, where an aged lady took three grains of a white powder, prescribed for her by her medical attendant. In about ten minutes afterwards, she was seized with coma, and died in the course of an hour. The medicine which she took was sulphate of quinine. In such a case it might have been most plausibly said,—morphia or some other poisonous alkaloid had been swallowed; but the circumstances were well known: death was due to apoplexy. In another instance, a woman, aged 37, rose in the morning in her usual health, with the exception of having a slight headach, and immediately after taking breakfast, was attacked with violent vomiting, which continued for half an hour, when she fell down and died suddenly. Here again there was room for suspecting poison, owing to the time of the occurrence of symptoms, but it was proved that the woman had died of disease of the brain. Many more such cases might be reported; but these will show that all inferences of poisoning under such circumstances should be drawn with caution.

The following case reported in the Medical Gazette, is of some interest in relation to the question which we are here considering:—A gentleman, suffering from what were supposed to be rheumatic pains, applied to an eminent London practitioner for advice. He prescribed for him draughts containing iodide of potassium in the proportion of six to eight grains to each dose. The draughts were prepared by the medical attendant, who administered one to the patient—the latter being in his usual health. He had scarcely swallowed it, when he complained of its being too strong, and declared that he was poisoned. A person present tasted a portion of the liquid, and said it had a very strong taste. The medical attendant put the other draughts into his pocket, saying he would dilute them, and left the house. In about half an hour the patient felt extremely ill. Two other medical men were sent for, but before they could arrive, probably an hour and a half had elapsed, and they found the patient dead. It is not stated under what symptoms deceased laboured; but it is loosely said that these created a strong suspicion of poisoning. The medical attendant who administered the draught was examined before the coroner, but nothing was elicited as to the cause of death. The remaining draughts were not ordered to be analysed, nor was any post-mortem examination of the body made. One medical witness was asked whether eight grains of iodide of potassium, or even twice that quantity, would be sufficient to cause death. He replied in the negative, and the jury returned a verdict of "Died by the visitation of God." The investigation of this case seems to have been so disgracefully conducted, that it is impossible to speculate on what could have been the cause of death,—whether poison or disease. The principal reason for suspecting poison was, that the symptoms came on soon after the taking of the medicine; but this, as it is well known, is not in itself evidence of poisoning. For any thing that appears in the report, the deceased may have been struck with fatal disease of the heart or brain, about the time of taking the medicine; and the symptoms preceding death, may have been due to a mere coincidence.

It seems highly probable that the mere fact of a person eating a full meal after *long fasting* will give rise to symptoms resembling those of irritant poisoning,—a circumstance which does not appear to have attracted the attention of writers on toxicology. Mr. Holland, of Manchester, has communicated to me two cases of this description. In one the symptoms were very violent, and the patient nearly died, in fact was laid out for dead. The other patient suffered from severe gastralgia for several weeks. Poisoning was at first strongly suspected, but the suspicion was removed by the fact, that others in health had partaken of the same food, principally potatoes mixed with gravy, without any injury; and there was no reason to suppose that any irritant poison could have been mixed with the food. The two who suffered, were extremely weak and exhausted from long fasting, and were observed to eat their food, which was quite wholesome, voraciously.

3. IN POISONING WHEN SEVERAL PARTAKE AT THE SAME TIME OF THE SAME FOOD OR MEDICINE (MIXED WITH POISON) ALL SUFFER FROM SIMILAR SYMPTOMS.—This character of poisoning cannot always be procured; but it furnishes good evidence of the fact when it exists. Thus, supposing after a meal made by several persons from the same dish, only one suffers, the suspicion of poisoning is considerably weakened. The poisoned article of food may be detected by observing whether they who suffer under symptoms of poisoning, have partaken of one particular solid or liquid in common. In a case of accidental poisoning at a dinner-party, a friend of mine observed that those who suffered from the symptoms, had taken port wine only: the contents of the bottle were examined, and found to be a saturated solution of arsenic in wine. In general, considerable reliance may be placed upon this character, because it is very improbable that any common cause of disease should suddenly attack with violent and alarming symptoms, many healthy persons at the same time, and within a short period after having partaken of food together. We must beware of supposing that where poison is really present, all will be attacked with precisely similar symptoms; because, as we have seen, there are many causes which may modify them. In general, that person who has partaken most freely of the poisoned dish will suffer most severely, but even this does not always follow. There is a well-known case recorded by Bonnet, where, among several persons who partook of a dish poisoned with arsenic, they who had eaten little and did not vomit, speedily died; while those, on the other hand, who had partaken largely of the dish, and had in consequence vomited freely, recovered.

It was just now remarked, that there is no disease likely to attack several healthy persons at the same time, and in the same manner. This is undoubtedly true, *as a general principle*, but the following case will show that mistakes may occasionally arise even under these circumstances. It occurred in London, during the prevalence of the malignant cholera in the year 1832. Four of the members of a family living in a state of great domestic unhappiness, sat down to dinner in apparently good health; some time after the meal, the father, mother, and daughter, were suddenly seized with violent vomiting and purging. The stools were tinged with blood, while the blueness of the skin, observed in cases of malignant cholera, was wanting. Two of the parties died. The son, who was known to have borne ill-will against his father and mother, and who suffered no symptoms on this occasion, was accused of having poisoned them. A strict investigation took place before the coroner; but it was clearly shown by the medical attendant, that the deceased persons had really died of the malignant cholera, and there was no reason whatever to suspect that any poison had been administered to them. In this instance, it will be perceived that symptoms resembling those of irritant poison appeared suddenly in several individuals in perfect health, and shortly after a meal. We hereby learn that the utility of any rules for investigating cases of poisoning, depends entirely on the judgment and discretion with which they are applied to particular cases.

Instances in which a whole family are simultaneously attacked with symptoms resembling those of poisoning, occasionally present themselves, and are often attended with great obscurity. The case of the Arzone family is in this respect of some interest. The family, consisting of the father, mother, and three children, were in good health up to the evening of December 30, 1840. On January 1, 1841, the father, a manufacturer of colours, was suddenly taken ill with griping pains and purging, which never ceased until death. He was sick at times, but never vomited; the motions were offensive and black; had frequently cold fits during the day, followed by much fever. His joints were swollen and painful. He died on the 20th January. On inspection, the stomach and intestines were found healthy. The lungs and pleura presented strong evidence of inflammation sufficient to account for death. The three children, as well as the mother, after suffering from somewhat similar symptoms, died,—the death of the mother being accelerated by parturition. All complained of general soreness of the fleshy parts of the joints, great sensibility of the skin, and pain produced by the least change of posture; they suffered from diarrhœa, complained of a cankery or metallic taste in the mouth, and there was a watery state of the mouth and eyes, with a dry cough. The abdominal viscera showed no change from the normal state, but the lungs were congested. (Med. Gaz. xxx. 326.) The symptoms could not be referred to any epidemic or malaria; the man, to my knowledge, lived in a healthy situation, and none of his neighbours were attacked. It was therefore difficult to ascribe them to malaria, or any disease depending on natural causes, occurring simultaneously in so many members of a family, previously healthy. On the other hand, there was no evidence that poison had been taken in the food, and the appearances in the body were not indicative of the action of any irritant, yet the symptoms and appearances were in some respects compatible with the hypothesis of chronic poisoning, either from the slow inhalation of the fine powder used in the preparation of colours, or from their introduction into food through want of cleanliness. There was no doubt that he employed the arsenical cobalt ore (the Tunaberg ore, see post, Arsenic) in the preparation of a kind of ultramarine; and possibly, as it was suggested at the time, impalpable dust in the preparation of this substance may have given rise to the symptoms. We shall see hereafter (Arsenite of Copper, Carbonate of Lead,) that these poisonous pigments may penetrate into the system in a most insidious manner, and produce the most serious effects. Walls painted with these colours, have thus caused alarming symptoms in those who inhabited the apartments. It is not at all improbable that the oil of turpentine, which is largely used in grinding colours, may by its volatility become the vehicle for the diffusion of the poisonous pigment.

The simultaneous occurrence of symptoms terminating fatally in two or more persons, is always well calculated to excite grave suspicion of poisoning; and a diagnosis can only be formed by noting the character of the symptoms, or, if this source of evidence be wanting, by the detection of poison in the food or bodies of the individuals. A simultaneous attack merely furnishes a presumption in favour of poisoning, to be supported or rebutted by other circumstances. A case which will be more particularly described hereafter (see CONVULSIONS,) was referred to me in December 1846, by Mr. Wood, coroner for Surrey, in which two young children, previously healthy, died under similar symptoms, very suddenly, and after a short illness. It was reasonably suspected in the first instance, that narcotic poison had been given to them; but an examination of the facts of the case, as well as an analysis of the food and contents of the stomachs, proved that poison was not the cause, and thus removed a heavy load of suspicion from the parents.

Obscure symptoms of poisoning may occur simultaneously in several members of a family from accidental causes, the nature of which may not be even suspected. Thus, various articles of food may become poisoned by copper through want of cleanliness in the use of culinary utensils (see COPPER;) or the water supplied to a house may be contaminated with lead from the use of that metal in pipes, cisterns, or merely as a cover to a tank (see CARBONATE OF LEAD.) The safety of the individuals, and probably the exculpation of an innocent person, wrongly accused of poisoning, will depend on the acumen of the practitioner in discovering the real cause. In reference to this question, the case of *Solomon* v. *Lawson*, tried at the Surrey Lent Assizes, 1845, is of some medico-legal interest. It was alleged that the defendant had libellously stated that the plaintiff, who was in the habit of supplying water to ships at St. Helena, had supplied some water poisoned with lead or copper to a particular ship, and that this was the cause of an illness which prevailed among the passengers on the homeward voyage. It appeared in evidence that five of the officers and the steward were affected, and two of the officers suffered severely from symptoms which, although slightly varying in the respective cases, the surgeon of the ship did not hesitate to refer to some metallic poison. Two medical men who attended three of the passengers on their return to England, declared that in their opinion the effects were due to lead, which was rendered probable by the fact that partial paralysis of the lower extremities and blueness of the gums were among the symptoms. Some of the crew, however, did not suffer, although they are stated to have used the same water. The water, as it was supposed but not proved, was analysed, and no lead or copper was found in it. As so much depended on this analysis, it is to be regretted that its identity was not more clearly made out, and that it was not assigned to some of the most eminent chemists in London. It is impossible, I think, to draw any other conclusion from the evidence, than that drawn by the medical witnesses, *i. e.* that the cause of the symptoms was really owing to some metallic poison, probably to lead. The jury, however, held, under the direction of the judge, that the water was not proved to have been poisoned, and returned a verdict, with very heavy damages, against the defendant. To have attained strict justice in this case, the facts, it appears to me, should have been referred to a medical board composed of experienced chemists and physicians. There is sufficient precedent for this, in the legally-recognised assistance afforded to the Admiralty courts on nautical questions, by the masters of the Trinity House.

It may be here proper to remark, that the water of wells in the neighbourhood of Chemical works is often impregnated with poison. Persons who unsuspectingly use this water may be attacked with symptoms of poisoning, and die from the effects. In the Registrar-General's Quarterly Report for 1846, it is stated that nearly the whole of the members of a family in Derbyshire, died from having drunk water impregnated with arsenic, which was drawn from a well contiguous to certain chemical works attached to the premises. (Med. Gaz. xxxvii. 843.)

It is well to bear in mind, in conducting these inquiries, that symptoms resembling those produced by irritant poison, may be sometimes due to the description of food which may have been taken at the meal. Besides flesh rendered unwholesome from disease and decay, there are certain kinds of shell-fish, as well as pork, bacon, sausages, cheese, and bread, which, under certain circumstances, may give rise to formidable symptoms, and even death. In such a case, all the foregoing characters of poisoning are brought out; and, indeed, the case may be regarded as one of poisoning by an animal or vegetable irritant. The diagnosis is difficult; and great ambiguity frequently arises, from the fact that not more than one or two individuals may be affected, who have frequently before partaken of the same kind of food without any particular inconvenience (see Animal Irritants.)

4. The discovery of poison in the food taken, or in the matters vomited.—One of the best proofs of poisoning, in the living subject, is the detection of poison by chemical analysis, either in the food taken by the person labouring under its effects, or in the matters vomited. The evidence is, of course, more satisfactory when the poison is discovered in the matters vomited, than in the food; because this will show that poison has really been taken, and will readily account for the symptoms. If thrown away, we must then examine the food of which the patient may have partaken. Should the results in both cases be negative, the probability is, that the symptoms may have been due to disease. In investigating a case of poisoning in the living subject, a medical jurist must remember, that poisoning is sometimes *feigned*, and at others, *imputed.* It is very easy for an artful person to put poison into food, and to accuse another of having administered it, as well as to introduce poison into the matters vomited or discharged from the bowels. There are few of these accusers who go so far as to swallow poison under such circumstances, there being a great dread of poisonous substances among the lower orders; and it will be at once apparent, that it would require a person well versed in toxicology, to feign a series of symptoms which would impose upon a practitioner at all acquainted with the subject. In short, the difficulty reduces itself to this:—What inference can we draw from the chemical detection of poison in food? All that a medical man can do, is to say whether poison be present or not in a particular article of food:—he must leave it to the authorities of the law, to develope the alleged attempt at administration;—but if the poison have been actually administered, then we should expect to have the usual symptoms. With regard to the detection of poison in the matters vomited from the stomach, this affords no decisive proof that it has been swallowed except under two circumstances:—1. When the accuser actually labours under the usual symptoms of poisoning, in which case there can be no feigning, and the question of imputation is a matter to be established by general evidence. 2. When the matters are actually vomited into a *clean vessel* in the presence of the medical attendant himself, or of some person on whose testimony perfect reliance can be placed. (For an atrocious case of imputed poisoning in which the accused party had a narrow escape of his life, see page 47.)

CHAPTER V.

EVIDENCE FROM THE NATURE OF THE SYMPTOMS—DISEASES RESEMBLING IRRITANT POISONING—CHOLERA—GASTRITIS — ENTERITIS — GASTRO-ENTERITIS—PERITONITIS —PERFORATION OF THE STOMACH—STRANGULATED HERNIA—INTERNAL STRANGULATION OF THE INTESTINES—DISEASES RESEMBLING NARCOTIC POISONING—APOPLEXY —SUDDEN DEATH FROM NATURAL CAUSES—POST-MORTEM EXAMINATIONS—EPILEPSY —TETANUS—CONVULSIONS IN INFANTS—DISEASES OF THE BRAIN AND SPINAL MARROW—OF THE HEART—DEATH FROM DISTENTION OF THE STOMACH—RUPTURES OF THE GALL-BLADDER.

NATURE OF THE SYMPTOMS.—One of the most important means of diagnosis upon which the physician relies, is the nature of the symptoms under which the patient is labouring. In cases of poisoning the symptoms are commonly well marked, and have a peculiar character; those of disease are less certain, and are more likely to create embarrassment. Owing to this, it happens that in practice, disease is much more liable to be mistaken for poisoning, than poisoning for disease. An account of the symptoms produced by the different classes of poisons will be found at page 38; and the special details,—in the description of each poison respectively. In this chapter it will therefore only be necessary to enumerate on the one hand those diseases, the symptoms of which might be mistaken for irritant poisoning, and on the other those which might be mistaken for narcotic poisoning.

DISEASES RESEMBLING IRRITANT POISONING.—The diseases, the symptoms of which resemble those produced by *irritant* poisons, are cholera, gastritis, enteritis, gastro-enteritis, peritonitis, perforation of the stomach or intestines, strangulated hernia, colic, and hæmatemesis.

CHOLERA.—It is necessary here to distinguish the common English cholera from the Asiatic or malignant form of the disease. In the ASIATIC CHOLERA there is usually sudden and extreme prostration of strength; the surface of the body is very cold, and sometimes has a dark livid or leaden hue, especially observed in the skin of the hands and feet; the breath is cold as it issues from the mouth; the matters discharged from the bowels are very copious, resembling rice-water with flakes of coagulated mucus floating in them. There is the most intense thirst, and the patient will drink large quantities of cold water. The symptoms of poisoning by arsenic and other irritants are wholly different from these, if we except perhaps the intense thirst which is present in both cases. The common ENGLISH CHOLERA, as it occurs in summer and autumn, closely resembles arsenical poisoning in its symptoms. Thus, an attack often comes on in a healthy subject in about half an hour after a meal. It is accompanied by vomiting and purging, and by violent pain in the abdomen, continuing until death when the case terminates fatally. It may usually be traced to some indigestible food of which the patient has partaken.

Many acquittals on criminal charges have taken place from the great difficulty which exists in distinguishing this last-mentioned form of cholera from arsenical poisoning; and, in truth, it may be observed, that if in any case medical evidence rested on symptoms alone, it would be scarcely possible, in some instances, to draw such a clear distinction between the symptoms of this disease and those of poisoning, as the law would deem absolutely necessary for conviction on a criminal charge. The rules recommended for forming a diagnosis, as they are laid down by the best writers on toxicology, do not appear to be very satisfactory. Perhaps the following may be taken as a statement of the most striking differences. In irritant poisoning the evacuations are often tinged with blood; in cholera they are not tinged with blood, but commonly deeply coloured by bile. In irritant poisoning, these evacuated liquids will sooner or later yield traces of poison when analysed. In cholera this is of course not the case. The attack of cholera is commonly dependent on some irregularity of diet, and appears chiefly in summer and autumn. Irritant poisoning may occur at any season. Except when it prevails in a severely epidemic form, from intense heat or other causes, and attacks the very aged or the very young, English cholera is not often fatal; and when it does prove fatal, it is commonly after three or four days from its commencement. In irritant (arsenical) poisoning, death is a common result in twenty-four hours, when the symptoms produced by the poison are such as to have resembled those of cholera, *i. e.* poisoning in its most acute form. In irritant poisoning, the symptoms usually come on in about half an hour or an hour after a meal; and although cholera may commence its attack at about the same period, yet, supposing several persons to have partaken of the food, all will suffer more or less if it be really a case of poisoning,—not if it be a case of cholera. It would be at least something very unusual, that several healthy persons should be attacked by cholera at the same time, unless the attack were owing to some improper kind of food used at the meal. (See case, p. 51.) Lastly, an analysis of the food may serve to determine whether irritant poison was or was not the cause of the symptoms. Of all irritant poisons, arsenic comes the nearest to cholera in the character of the symptoms. It is right to bear in mind, however, that a case of arsenical poisoning is often accompanied by special symptoms which are met with neither in cholera nor in any disease resembling it. Thus in persons who have taken arsenic and survived the first effects of the poison,—the conjunctivæ of the eyes often become inflamed, sometimes at a very early period,—there is also great irritation of the skin, followed by a peculiar herpetic eruption;—and occasionally paralysis and coma appear among the symptoms. In cholera, nothing of the kind is witnessed; hence we have in these peculiarities, means for assisting us in our diagnosis. When the person dies, a post-mortem examination, with an analysis of the contents of the stomach, or if death speedily follow the attack, an analysis of the tissues of the soft organs, will often remove any doubts that may have existed on the real nature of the case. In several recent cases, arsenical poisoning has been mistaken for cholera, and the fact of poisoning remained concealed until an analysis was made. (See cases of *Reg.* v. *Chesham*, Essex Lent Assizes, 1847; and *Reg.* v. *Foster*, Suffolk Lent Assizes, 1847.)

Gastritis, Enteritis, Gastro-enteritis, Peritonitis.—These diseases do not commonly occur without some obvious cause; indeed, the two first, in the acute form, must be regarded as the direct results of irritant poisoning. Thus arsenic and other irritants, when they prove fatal, commonly give rise to inflammation of the stomach and bowels. In all cases where these diseases present themselves, the object of the practitioner is therefore to determine the *cause* of the inflammation, whether it be due to natural changes, or the action of an irritant poison. The diagnosis will chiefly rest, 1, Upon the time of the occurrence of the symptoms after a meal. 2. The order of their occurrence. 3. The obstinate constipation of the bowels, which is observed in gastritis and enteritis, is contrasted with the violent vomiting and purging met with in irritant poisoning. 4. The presence of fever in these diseases. The history of the case so clearly explains its nature, that we seldom hear of these diseases being mistaken for irritant poisoning. The same observations apply to peritonitis, in which disease there is also constipation, and but little vomiting. It has been doubted by some pathologists whether the diseases above mentioned can occur spontaneously, and without any apparent cause. All agree that instances of idiopathic acute gastritis are rarely observed in individuals otherwise healthy. Two cases however were reported to the Medico-Chirurgical Society, by Dr. Burne (Med. Gaz. xxv. 414,) and another case has more recently occurred to Mr. Berncastle (Lancet, March 1844.) The symptoms were of the usual character—constant vomiting, no diarrhœa, and rapid sinking. After death the stomach was found in a high state of inflammation, but all the other viscera were healthy. A suspicion of poisoning did not attach to the case. Acute enteritis from natural causes is much more common than acute gastritis. These diseases, in a chronic form, have a very slow course, and may be a secondary result of irritant poisoning. The symptoms are unlike those produced in the acute form of poisoning.

Perforation of the stomach and intestines.—The symptoms attending perforation of the stomach, in some respects resemble those of irritant poisoning. They often occur suddenly to a healthy person after a meal. This disease is almost invariably fatal, and may be immediately recognised on a post-mortem examination. Even in the rare cases in which it is not fatal, the means of diagnosis are not difficult. (See post, Ch. ix.)

Strangulated hernia.—It is difficult to suppose that this disease should ever be confounded with irritant poisoning. The seat of pain, with an examination of the part, would at once show the physical cause to which the symptoms were due.

Ileus, Internal Strangulation.—It may happen that there is no outward tumor, but that symptoms commence suddenly in a previously healthy person, and death takes place from strangulation internally. A report of an interesting case of this kind was read by Dr. Snow before the Med.-Chir. Society, in June 1846. The patient died in four days, and on inspection, a portion of the ileum was found strangulated in an aperture of the mesentery (Med. Gaz. xxxviii. 1049.) Two cases of a similar kind are quoted by Dr. Snow in his paper. In the same number of that journal, is reported a fatal case in which the cause of strangulation was produced by the weight of a supplementary spleen drawing the omentum into a cord (p. 1053.) Other cases in which there were various mechanical causes of internal strangulation are reported at pp. 1073 and 1075. The symptoms of this disease are a very sudden access of severe pain, chiefly confined to one spot, not in the region of the stomach, as in irritant poisoning, but in the central or lower part of the abdomen—severe and constant vomiting, not always of fæcal matter, but in some instances of a yellow or green-coloured liquid. There is, of course, obstinate constipation, if we except what may be discharged from the lower bowel. The diagnosis is commonly not difficult, and a careful post-mortem inspection will immediately lead to the detection of the cause of death. The case is in general fatal, as there are no means of relieving the strangulation: hence post-mortem evidence is rarely deficient. In the following case (that of a friend) in which the cause of the symptom was very obscure, the suspicion of poisoning was, in the first instance, strong. This gentleman, while in good health, was suddenly seized, two hours and a half after his dinner (taken as usual) with the most severe pain in the epigastrium. This was succeeded by nausea and vomiting, which continued without intermission for some hours. Twenty-four hours after the attack he was seen by a medical man: the vomiting had ceased, but every thing which he swallowed was rejected. There were frequent rigors; the pulse quick and wiry; extremities cold; face Hippocratic; voice feeble; and a cold sweat covered the body. The epigastrium was tender on pressure, and there was a fixed pain, increased by pressure, radiating from the cæcum along the ascending colon. There was no hernia; but the abdomen was hard, and drawn spasmodically towards the vertebral column, as in cases of colico pictonum. The tongue was dry, coated with a brown fur; the lips parched; and there was intense thirst. As there had been no motion for twenty-four hours, purgatives were administered, which produced evacuations; and under this and other treatment the patient speedily recovered.

That the symptoms in this case were not due to poison was established, 1, by their very sudden invasion in a severe form; 2, by the time which had elapsed since any food had been taken; 3, by the fact that other persons partook of the same food, and did not suffer. The symptoms were unlike those produced by arsenic or the common irritants; but they in many respects resembled those observed in the reported cases of ileus depending on mechanical strangulation of the intestines. The obstruction may have been caused by spasmodic constriction of the intestines; for if the cause had been mechanical, it is difficult to understand how it could have been removed, or how the purgative should have acted so freely.

Cases of obstructed intestine have occasionally given rise to medico-legal inquiries. The following is quoted by Flandin. A German physician was required to inspect the body of a merchant who had lived upon bad terms with his wife. It was supposed that she had killed him by poison. The deceased, it appears, had been confined to his bed for several days, and had complained of incessant nausea, vomiting, and severe pain. A careful post-mortem inspection of the body showed that a portion of the colon had become strangulated, and that it was in a gangrenous condition. The accused was immediately discharged. (Des Poisons, i. 295.) This fact with the one which follows, proves that the abdominal viscera ought to undergo a strict examination in suspected death from poison. In February 1829, an opera-dancer was suddenly seized with violent vomiting and obstinate constipation. There was but little pain in the abdomen, and no thirst. After two days there was a fixed pain in the right iliac region; the vomited matters were of a yellow colour, and soon assumed a fæcal character. The existence of ileus was suspected, and the case soon terminated fatally. Some days after the interment of the body reports were spread to the prejudice of the husband. The body was disinterred, examined, and a statement made that deceased had died from a chronic *gastro-enteritis*. The legal authorities then required MM. Orfila and Rostan to make an inspection; and they found that the colon was strangulated near its junction with the cæcum by a short fatty appendage, adhering at its two ends to the mesentery, so as to form a kind of ring. There was no trace of poison. Strangulation was obviously the cause of death. (Orfila, Toxicologie, ii. 720, 1843.)

The following case shows that fatal strangulation may take place even before the protrusion of a hernia. A soldier, who had hitherto enjoyed good health, was suddenly seized with violent pain in the abdomen, vomiting, and obstinate constipation. He was taken to a military hospital. Severe pain was experienced on touching the parietes of the abdomen, especially in the region of the umbilicus. There was no appearance of a tumour in any part. The pulse was small and frequent, and there was intense thirst. Nine hours after admission, there was copious vomiting, with some appearance of fæcal matter, and the man died immediately afterwards. On inspection, a portion of the ilium, about the size of a small nut, was found strongly compressed in an aperture in the right inguinal canal; but there was no accumulation of fæcal matter in or near the part. The symptoms and appearances rendered it certain that deceased had died from peritonitis (Gazette Médicale, Dec. 19, 1846, p. 995.)

An examination of the parietes of the abdomen, may not always suffice to indicate the cause of the sudden illness and death. Nevertheless, the obstinate constipation, with other symptoms, will in general be sufficient to show that they cannot be ascribed to irritant poison. In all of these doubtful cases, if the symptoms be really dependent on poison, some connexion may be established between the last meal taken and the period of their occurrence.

Colic.—This disease can only be confounded with one variety of irritant poisoning, namely, that induced by the salts of lead. But it is to be observed, that the poisonous salts of lead are very rarely used criminally, and when they are taken in sufficiently large doses to kill rapidly, the symptoms resembling colic are mixed up with those of irritant poisoning,—so as to render it impossible for a practitioner to refer them to that disease alone.

Hæmatemesis.—In this disease, there is neither pain nor diarrhœa; and there is a copious discharge of blood by vomiting. These characters show that it cannot be easily mistaken for irritant poisoning.

Diseases resembling narcotic poisoning.—We may now proceed to speak of the diseases which have symptoms resembling those induced by *narcotic* poisons. They are apoplexy, epilepsy, diseases of the brain, diseases of the heart, and rupture or distension of the stomach. Indeed, it may be remarked, that every condition of the body in which life is liable to be suddenly destroyed, from whatever cause, may be mistaken for narcotic poisoning. The various causes of sudden death should therefore be especially studied by a medical jurist. These are not very numerous, and are principally confined to disease affecting the brain, heart, and lungs. For an account of these causes, I must refer the reader to the Ann. d'Hyg. 1838, ii. 145; 1843, ii. 435. There is another point to be attended to, namely, that those fatal diseases only of those important organs, are likely to be confounded with this form of poisoning, the existence of which had not been previously suspected or announced by the usual attendant symptoms. On the trial of *Tawell* for poisoning Sarah Hart by prussic acid, the statistics of the causes of sudden death were entered into in the defence, in order to establish a probability that the deceased had died from natural causes, among which was placed "mental emotion!" It may be as well therefore to state, that on an average of five years, 1838–42, the annual number of sudden deaths in England and Wales amounted to 3600, or one in one hundred and thirty-eight of the total deaths. (Registrar-General's Report, 1843.) Although inquests are held in all of these cases, for the alleged purpose of determining the cause of death, no cause whatever is assigned in *two-thirds* of the number: and thus a vast source of knowledge of great importance in the settlement of a disputed case of poisoning, is entirely cut off from the profession!

Apoplexy.—Narcotic poisons, of which we may take opium as the type, actually seem to produce this diseased condition of the brain. The distinction of apoplexy dependent on disease, from that kind of apoplexy induced by poison, is extremely difficult unless we can obtain a full history of the case. The following circumstances may be remembered in our diagnosis. 1. Apoplexy, as a disease, is sometimes preceded by warning symptoms before the fatal attack comes on. In poisoning, such symptoms would be wanting unless the poison were administered to a person who had already been threatened with apoplexy. 2. Apoplexy, as a disease does not commonly attack persons under the age of thirty. The fatal cases increase progressively with age, and according to the researches of Dr. Burrows, the disease is most common between the ages of sixty and seventy. We shall presently see that, there are, however, exceptions to this rule. Poisoning may be witnessed in a person at any age. 3. The relation between the time of the attack, and the time at which food or medicine was last taken. Thus if the comatose symptoms do not come on until five or six hours after some liquid or solid has been swallowed, they are much more likely to be dependent on apoplexy from disease than on poison. This is a most important character; but its occurrence is of course purely accidental, for it is by no means unusual that an attack of apoplexy should speedily follow a meal made by a previously healthy person. However, several cases have already been related, which show that this may be sometimes usefully employed to distinguish disease from poisoning (ante, p. 45.) 4. In apoplexy from disease, it is usually observed that coma is at once induced:—but in poisoning, coma comes on slowly, and is generally preceded by vertigo and stupor. 5. The discovery of poison in the food taken or in the contents of the stomach:—this would at once establish the fact of poisoning. 6. The discovery of appearances in the brain indicative of apoplexy, such as effusion of blood. This would negative, cæteris paribus, the presumption of poisoning.

It is to be observed, that in all cases of disease simulating narcotic poisoning, the disease is assumed to prove fatal:—hence there is always the opportunity of searching for the two last-mentioned characters. We do not hear of an attack of apoplexy from which a person recovers, ever being mistaken for a case of poisoning by opium, but we hear of poisoning by opium being not unfrequently mistaken for apoplexy or convulsions. Dr. Birt Davies has published the two following cases. A person died in what was considered by the physician and surgeon attending to be a fit: but opium was found in the stomach. A person was attended by a physician and surgeon for some hours. The illness and death were ascribed to and treated by them for apoplexy, but it was proved beyond all doubt that the deceased died from laudanum. (Borough Inquests. Birmingham, 1845.) Such cases I am persuaded are frequent, having had several referred to me for examination during the last few years. Cases have of late been unfortunately too common in which deaths have been registered as from "natural causes," when on an exhumation of the bodies some weeks or years afterwards, the deceased have been found to have died from poison. It is impossible to say how many of such cases escape notice for one which is brought to light. These facts show that inquests without post-mortem examinations, in many instances serve, by lulling suspicion, to conceal rather than to detect crime.

A case was lately tried at the Lincoln assizes, which shows that a crafty criminal may easily deceive a medical practitioner, and that the coroner's inquest, as it is at present conducted, is not fitted to detect these secret cases of poisoning. In this instance a confession was made; but how many instances escape detection for want of a confession on the part of a criminal, it is impossible to conjecture. An inspection of a body is not required by many coroners unless there are strong circumstances for suspicion in the shape of public rumour; but in respect to criminals, who have well-calculated their plans, these circumstances are not likely to come to light except from a post-mortem inspection, and an analysis of the contents of the viscera. It does not appear that an inquisition was held, or inspection made, in the case alluded to, until some time after the bodies of the deceased had been interred, and then it was too late. A woman was charged with the murder of three children, by poisoning one of them with arsenic, and the other two with opium. She pleaded guilty, and confessed the manner in which the crime was perpetrated. She had succeeded in poisoning two of the children without being detected; although suspicion was so strong that she was tried, but acquitted, at the previous assizes, on the charge of having poisoned one of them. In the third case, she admitted having secretly given to the deceased (her own infant,) about three weeks old, a tea-spoonful of laudanum. The child was soon afterwards seized with convulsions; a medical practitioner was sent for, who, deceived by the statement of the woman, treated it as a case of ordinary convulsions in children, and ordered a warm bath. The child died in about twenty hours, continuing, according to the prisoner's statement, in convulsions during the greater part of that time. No suspicion appears to have been entertained of the real cause of death, and the case would probably have remained undiscovered, but for the prisoner's confession. It is remarkable that this child survived so long; the woman, however, prevaricated as to the quantity of laudanum which she gave it, therefore it is difficult to draw any conclusion from her statement, except that the deceased was actually poisoned by opium. (The *Queen* v. *Joyce*. Lincoln Aut. Ass. 1844.) In one case referred to me (Sept. 1844,) the jury, under the direction of a coroner, returned a verdict of death from poison ("misadventure,") while the stomach of the deceased was in my custody, and before it had even been opened, or the seals of the vessels containing it had been broken! In another, in which there was the very strongest reason to suspect death from poison administered by a quack, the coroner and jury declined waiting for an analysis of the contents of the stomach, although strongly advised by the medical witness who inspected the body,—and returned a verdict of "*natural death.*" (See also the case of *Reg.* v. *Freeman*, Taunton Lent Assizes, 1845.) The Registrar-General has stated that in *sixty-six* out of every hundred inquests in cases of sudden death, no cause of death is assigned by the jury. Frequent verdicts of natural death, without an inspection of the bodies, tend to encourage the crime of secret poisoning, which appears to be rapidly spreading throughout this country.

In reference to the age at which apoplexy may make its attack, it may be remarked that healthy girls of the respective ages of sixteen and twenty-two, have died suddenly from this disease. There had been no warning symptoms whatever. In January 1839, a gentleman aged twenty-two years, retired to his bedroom in good health. Shortly afterwards a servant entered the room, and found him lying on the floor dead. On a post-mortem examination it was found that one of the vessels of the brain had become ruptured, and that a large quantity of blood had been effused on the surface. There was no doubt that this was a case of apoplexy in a young man who had suffered from no warning symptoms. It turned out on inquiry, that the father and mother of the deceased had both died suddenly from the same disease.

In the following case the suspicion of poisoning was actually raised, and was only removed by a proper medico-legal examination. In September 1838, a young healthy female, while sitting with her parents taking her supper, suddenly fell back in her chair in a state of insensibility. Medical assistance was called in, but she died in about eleven hours without recovering her consciousness. The parents were accused by the neighbours of having administered poison to the deceased at her supper: but the medical attendant, on making an examination of the body, clearly showed that death was caused by an effusion of blood on the brain, from the rupture of a diseased blood-vessel. As apoplexy is very unusual in the young, this rather tended to strengthen the suspicion of poisoning; although it is obvious that there is no common poison which would produce immediate insensibility except hydrocyanic acid; but when taken in so large a dose as to produce this sudden and violent effect, the probability is, that the patient would die in a few minutes. No poison was discovered in the body: death was undoubtedly caused by apoplexy.

I have known a child between two and three years of age, die from congestive apoplexy; and the disease has been observed to occur even in infants. Dr. A. Campbell reports a case of apoplexy proving fatal in a child only eleven days old. (North. Jour. Med., Jan., 1845.) A remarkable case, involving the question—whether death was caused by prussic acid or apoplexy, came before the Senate of Chambery in April 1843. I allude to that of *M. Pralet* (Ann. d'Hyg. xxvi. 399; xxix. 103, 474,) which appears to have excited as much attention on the continent, as the case of Sir T. Boughton in England. Several medical witnesses deposed that the deceased had died from prussic acid, administered to him by M. L'Héritier, the accused. Orfila was required to examine the medical evidence, and found it extremely defective. The inferences drawn from the application of the chemical tests were highly improper; and the results were essentially negative. Had it not been for the interference of Orfila, it is most probable that the accused would have been convicted, more from the strong medical opinions against him, than from the medical facts of the case. The witnesses appear to have acted on the principle, that the whole of their duty consisted in rendering the charge of poisoning probable; whereas, we shall hereafter see that no person can be convicted of this crime on mere *probability:* the fact of poisoning must be made reasonably certain, either by medical or moral evidence, or by both combined. The case of *Tawell* presents a converse illustration. Here, where the cause of death was obviously prussic acid, a struggle was made to show, on the most untenable hyper-chemical and hyper-pathological grounds, that the deceased had died from apoplexy or some other sudden cause. (Bucks Lent Assizes, 1845.)

It is highly important for the medical jurist to bear in mind, that a superficial examination of the body will not suffice to reveal the cause of death on these occasions. The fatal lesion may be in or near the pons Varolii or the cerebellum. Dr. Charles Bell has reported a case, which is in this respect instructive. A stout, strong, and hale labourer, aged sixty-two years, who had never complained of any indisposition, resumed his work as usual after having eaten a hearty dinner, and shortly afterwards, while wheeling a barrow, he dropped down senseless, and died in a few seconds. On a careful examination of the head, it was found that fatal effusion had taken place from a slight rupture in the right lateral sinus, midway between the torcular Hierophili and the jugular fossa. (Med. Gaz. xxxix. 31.)

In the following case there was a strong suspicion that the deceased had been poisoned by some companions with whom he had been drinking.

The deceased, a fisherman, had been drinking at a beer-shop with three or four friends, who took him home in an insensible state about two o'clock in the morning, threw him on the floor of the house, and left him. Two hours afterwards he was discovered by a relative, who supposed him to be intoxicated: he was then lying asleep, and breathing hard. He lay in this insensible state for two days, when a medical man was sent for, and he was found to be dead. He had vomited over the floor of the room a very dark-looking tarry kind of matter—some of which was found in the mouth. By order of the coroner the body was inspected the following day. The chief appearances were adhesions about the dura mater; injection of the pia mater, especially about the convolutions. There was no effusion of blood, but there was general congestion of the substance of the brain and its vessels. The stomach was removed and, with its contents, brought to me for examination. The contents of the organ were of a very dark colour, exactly resembling the appearance often seen in poisoning by sulphuric or oxalic acid. They were mixed with large quantities of mucus, and had a peculiar odour, somewhat resembling, as it was thought, that of blacking. A suspicion had arisen that his companions might have given him blacking to drink while he was in a state of intoxication. The dark viscid liquid scarcely possessed any acid reaction. The mucous membrane of the stomach itself was much corrugated. There was strong vascular injection throughout, but especially towards the cardiac extremity: a quantity of dark viscid matter was here closely adherent to the surface. There was no appearance of corrosion or chemical action on any part of the mucous membrane. The contents of the stomach were boiled with water, when they became coagulated, and of a much lighter colour. A turbid opaque liquid was obtained on filtration, which indicated only that degree of feeble acidity which is generally met with in the stomach; and the result of numerous experiments was, that there was no trace of sulphuric acid, nor of any other poison, in the organ or its contents.

Considering the nature of the symptoms, which were not those of irritant poisoning,—the post-mortem appearances, which were somewhat ambiguous, although they did not indicate corrosive poison,—and the chemical analysis, which clearly negatived the presence of poison, an opinion was given that the man had died from apoplexy brought on by excessive drinking, and that the dark matter found in the stomach, was nothing more than the altered hæmatosine of blood poured out with the mucus, probably as a result of the irritation produced by the alcoholic liquid taken. There was no smell to indicate the presence of any alcohol in the contents, at the time they were submitted to analysis.

Epilepsy.—This disease, in some of its symptoms, resembles poisoning by prussic acid only. If the symptoms depend on poison, some liquid or substance must have been taken immediately before their occurrence. If, however, nothing has been taken, the inference would be that the symptoms most probably depended on disease. Death is commonly very rapid in poisoning by prussic acid; but a first attack of epilepsy is not often fatal. If the person has suffered from previous attacks, the probability is, cæteris paribus, that the symptoms depend on disease. But epilepsy may by coincidence immediately follow the administration of a draught or the taking of food:—an analysis of the substance taken, would in such a case remove any doubt. Supposing none of this to be procurable, then we must remember, that epilepsy only simulates narcotic poisoning when the attack is rapidly fatal. Therefore, an opportunity will always present itself for verifying or rebutting the suspicion of poisoning, by examining the contents of the stomach. I have never met with an instance, where a case of epilepsy was mistaken for one of narcotic poisoning.

Tetanus.—This disease, when it occurs can generally be traced to some cause,—a wound, or other injury, involving tendinous or nervous structures. It may, however, arise from causes of a very simple kind, and independently of wounds,—as from exposure to wet and cold, or to a current of air. It may even come on without any apparent cause. Idiopathic tetanus is by no means common: it manifests itself by trismus (locked jaw,) opisthotonos, or emprosthotonos. The disease sometimes occurs spontaneously in young infants, within the first eight or ten days from birth (*trismus nascentium.*) Male adults, especially those who are of a robust and vigorous frame, are most liable to attacks of tetanus. According to Dr. Gregory, tetanus from cold occurs, for the most part, within three or four days after exposure to the exciting cause; while traumatic tetanus (from wounds) generally appears about the eighth day. (Practice of Physic, 378.) Other observers have found that tetanus from wounds very commonly shows itself from the fourth to the sixth day after the injury. The sooner it commences after an injury, the more rapidly fatal is its course.

Tetanus, or rather tetanic convulsions, may be produced by certain narcotico-irritant poisons, especially those belonging to the strychnos tribe,—as nux-vomica—strychnia, and all its saline combinations; and there is not only a strong similarity in the symptoms, but a post-mortem examination does not indicate the existence of any well-marked morbid changes in either case. In tetanus from disease or injury, there is a gradual progression of the symptoms. The rigid contraction commences in the masseter muscles; it extends to the fauces, back of the neck, and, lastly, descends to the abdomen and lower extremities. Professor Colles has remarked, that the muscles of the fingers are the last and least affected. (Lectures on Surgery, i. 72.) The muscular rigidity continues more or less throughout; whereas, in tetanus from poisoning, the spasm is more general, and there are interruptions or intervals of relaxation. A diagnosis will commonly rest upon the following circumstances:—1. The period of time which has elapsed since any substance, liquid or solid, was swallowed by the patient. 2. The gradual or sudden and violent accession of symptoms,—the latter indicating poisoning. 3. The duration of the case. Tetanus, as a result of injuries, rarely proves fatal in less than twenty-four hours; and in the idiopathic form, it seldom destroys life in less than three or four days. In tetanus, produced by compounds of strychnia given in fatal doses, the person rarely survives two hours. 4. The discovery of nux-vomica, strychnia, or other poison in the food, in the matter vomited, or in the contents of the stomach after death. Prussic acid gives rise occasionally to tetanic convulsions, manifested by opisthotonos, emprosthotonos, and general spasm of the trunk and extremities; but the very sudden invasion of symptoms after a liquid has been taken, the rapid termination of the case, and the presence of the poison in the stomach, will render a diagnosis easy.

CONVULSIONS.—This is a very frequent cause of death among young children. According to the Seventh Report of the Registrar-General (1846,) the yearly average of deaths ascribed to this cause amounts to 25,000. Convulsions (or fits, as they are vulgarly termed) may arise from the action of poison, especially of those belonging to the narcotic class, or from natural causes of disease. As they sometimes suddenly attack children, and prove fatal rapidly, a suspicion may arise that death has been caused by some poison administered to the child. Many cases of this kind have been referred to me of late years for investigation; and, from the frequency of their occurrence, and the unjust suspicions to which they may give rise, it is the duty of the practitioner to make himself acquainted with the common causes of this disease. Systematic writers have divided convulsions into symptomatic and idiopathic. They are most commonly symptomatic, *i. e.* depending on some disease or morbid condition of the system,—such as dentition, repelled cutaneous eruptions, hydrocephalus, exposure to cold, indigestion, worms, accumulation of fæces, improper food, or overdistention of the stomach and bowels with food; even a peculiar condition of the nurse's milk may become a cause. The younger and more irritable the child, the greater is its liability to an attack; and in these cases, the slightest cause of irritation to the nervous system may lead to it. About the time of the appearance of the first set of teeth, *i. e.* from the fifth to the eighteenth month, children are considered to be most liable to the disease.

When the convulsions cannot be traced to any of the causes above assigned, they are described as idiopathic, and are commonly referred to some primary disease of the brain; and this organ, after death, may be found in a state of congestion. Idiopathic convulsions sometimes run through their course and cause death very rapidly; but it is not at all improbable that, by diligent inquiry, some cause might be found. Dr. Underwood met with several cases where fine healthy children died suddenly from convulsions immediately after they had been overfed by their nurses.

Convulsions are a very common effect of the action of overdoses of opium on young children; and they are not to be distinguished from those which arise from natural causes. During the fit, the eyes are distorted, the pupils contracted or dilated. The spasm may affect the organs of respiration: the jaws are closed, and saliva, in a frothy state, escapes at the mouth. There may be also stertorous breathing; and, from impeded respiration, the tongue, face, and the surface of the skin will become livid owing to imperfect aeration of the blood, and the child may die asphyxiated. Under prompt and appropriate treatment, except when it depends on poison unsuspected, the attack may be alleviated, and the child recover. When narcotic poison is the cause, it will be found that some substance, either liquid or solid, has been given to the child not long before; and the treatment to be pursued in a suspicious case must be directed to its removal. (See OPIUM.)

Except by a chemical analysis of the food and the contents of the stomach, it is by no means easy to form a diagnosis. In a recent case (December 1846,) a suspicion arose that two children had been poisoned, from the singular fact that they died within a very short period of each other. T. R., an infant, aged seven months, was found by the mother, at 6 A. M., in a fit. It was livid in the face, frothing at the mouth, its limbs drawn up and rigid. She immediately took it to a person living in the same house, but it remained insensible until it died, about two hours afterwards. The child appeared well when put to bed the previous night, and had had its last meal (boiled bread and milk) about seven o'clock. The chief appearances, on inspection, were congestion of the brain and lungs; there was slight redness of the stomach. On the same morning, and about the same time, the other child, aged fifteen months, was found by the mother insensible, dark in the face, and struggling for breath. This child died in five minutes after it was found. On inspection, the only appearance was general congestion of the brain.

The stomachs and their contents, as well as a portion of the food given to the children the night before, were examined for opium as well as other poisons which were likely to have occasioned the symptoms: but no trace of poison could be discovered; and the contents of one stomach acquired a deep blue colour with a solution of iodine, bearing out the statement that bread had been given at the meal. There was no moral evidence to show that poison had been given; none was detected in the food; and had it been given by the mother, who found the children dying early in the morning, the probability is, that as there had been no vomiting, and death was rapid, the poison would have certainly been discovered by the odour, or in the stomach or its contents, by the usual tests. The opinion given was, that death had resulted from convulsions, probably produced by a congested state of the brain. The most remarkable feature in this case was the coincidence in seizure and the time of death; and, but for the good character of the parents and the results of a chemical examination of the food and the viscera, it would have been difficult to have satisfied the neighbours, that the children had not been destroyed by poison. The jury returned a verdict of death from natural causes.

The importance of not relying too strongly upon a coincidence in the attack of several persons labouring under similar symptoms following a meal, has already been pointed out. (See ante, p. 51.)

DISEASES OF THE BRAIN AND SPINAL MARROW.—Among these diseases, may be mentioned inflammation of the brain and its membranes, hypertrophy, and the formation of tumours. Such diseases are of a very insidious nature:—they sometimes give no warning of their presence, until the person, who may be in his usual health, is suddenly seized with stupor, followed by coma, and rapidly dies. All such cases resemble poisoning by opium: they can only be distinguished by the discovery of the affirmative characters of disease, on a post-mortem examination, and an absence of poison from the stomach. But the period of access of the symptoms after a meal, and the rapidity of death, will, in many instances, allow a practitioner to form a satisfactory diagnosis. I have already referred to a case, (ante, p. 49,) where a woman, aged 87, died suddenly, soon after having taken her breakfast. On examination of the body, there was found effused within the cranium a large quantity of bloody serum; and the brain and its membranes were much congested with blood. No poison was discovered in the stomach, and it is certain, that had death been due to a narcotic, some traces of it would have been found, in consequence of the great rapidity with which the deceased died. The only points in which this resembled a case of poisoning, were in the invasion of the symptoms soon after a meal, and their rapidly fatal termination.

DISEASES OF THE HEART.—The heart is subject to many diseases, which present the same insidious characters, as those of the brain. Thus they may remain for a long time latent, and then suddenly destroy life. They are only likely to be confounded with poisoning by prussic acid, owing to the rapidity with which death takes place. In all these cases, therefore, if the fatal attack occur suddenly some hours after food or medicine has been taken, there can be no reason for attributing it to poison. It is only when by a coincidence the symptoms appear at or immediately after something has been swallowed by the patient, that any doubt of the cause to which they may be due, can arise; and here, the doubt would be speedily removed by a post-mortem examination of the body. We must not expect, however, that in these fatal affections of the heart, well-marked post-mortem appearances will be always met with. Some pathologists have described a singular condition of this organ, under which the person dies suddenly after experiencing nausea, vertigo, and fainting. In such cases, the parietes of the heart have been found only preternaturally flaccid, and its cavities empty. This has been called by Mr. Chevalier, idiopathic asphyxia, and others have termed it syncopal asphyxia. It does not appear to be very common, for very little is known concerning it, or on what the cause of death really depends. In regard to its diagnosis in suspected cases, all we can say is, that if poisoning be not clearly negatived by concomitant circumstances, its usual affirmative characters are entirely wanting.

The question whether death has arisen from poison or from malformation or disease of the heart, occasionally presents itself to the medical jurist. The diagnosis is of some importance, since the local changes in the organ may be slight and easily overlooked. In July 1844, a woman, aged 45, fell suddenly in the street near Guy's Hospital, and died, gasping and pallid without being at all convulsed, in ten minutes after she had been brought into one of the wards. It was ascertained that she was a woman of irregular and intemperate habits, and that about an hour before, she had purchased a drachm of powdered nux-vomica at a druggist's. A paper containing the poison was found in her possession. There was no certain evidence that she had taken any of it, but unfortunately the quantity found upon her was not weighed. It was considered, however, by those who saw it, to be equal to about a drachm, the quantity purchased. On the report that poison had been taken, the stomach-pump was used; and a decoction of cinchona administered. On inspection eighteen hours after death, it was remarked that the lips and ends of the fingers were extremely blue. The heart was enlarged, the right cavities being very capacious, and distended with dark semifluid coagula. There appeared to have been originally two communications between the auricles. The pulmonary artery was of extraordinary width, while the aorta was narrow. The lungs were highly emphysematous and congested; the liver congested; the mucous membrane of the stomach slightly corrugated, and it presented a few spots of ecchymosis, which were probably produced by the stomach-pump. I examined the stomach and its contents, but no traces of nux-vomica or any other poisonous matter was found. Although it is possible that some of the poison might have been taken, and afterwards removed by vomiting or the stomach-pump, the symptoms and the short duration of the case showed that death could not be referred to it. There was no doubt that this was caused by a delay to the passage of the blood through the right cavities of the heart, from congenital malformation of the organ. It was ascertained that the deceased had been always liable to shortness of breath, and that she became blue upon any unusual exertion. (See Medical Gazette, xxxvi. p. 19.)

In January 1846, I was consulted by my friend, Mr. J. G. French, respecting a case to which some suspicion was attached. A man, aged 37, who had been for some time ailing and in low spirits, went out on his avocations, after having taken his breakfast as usual and was observed to fall suddenly in the street. This was between three and four hours after he had taken his breakfast. He was carried into a shop: some stimulant was given to him, but he died before Mr. French could arrive, which was about ten minutes after the seizure. It was ascertained that he had been kept in the erect posture after the attack. In order to remove suspicion of poisoning, the body was examined. The chief appearances were great enlargement of the heart, with a thinning of the parietes of the left ventricle. The mucous membrane of the stomach was slightly reddened about the lesser curvature and the cardiac orifice: at this part two small patches of ulceration existed in the membrane,—the ulcers being slightly thickened around the borders. Towards the pylorus the mucous glands were much enlarged. The contents, which amounted to about six drachms of a brown-coloured liquid, had a slight alkaline reaction and a faint aromatic odour. On standing, a slight sediment was deposited, but there was no solid or undigested matter; no appearance of mineral substance; no effused blood; and but little mucus. The kidneys were enlarged, and congested with venous blood. The liver was healthy; but the gall-bladder was distended, and contained twenty gall-stones. The results of a chemical examination were—1, That the liquid of the stomach contained no trace of poison, mineral or vegetable; 2, That it consisted of digested food, mucus, with a small quantity of sal-volatile and traces of common salt. Considering that the severe symptoms commenced long after any article of food had been taken, and that, had they arisen from any poison subsequently taken, it must, from the rapidity of death and the absence of vomiting, have been found in the stomach, it was inferred that death had not been caused by poison. The opinion given at the inquest was, that death had taken place from a diseased condition of the heart, producing fatal syncope.

DISTENTION OF THE STOMACH.—This is by no means an unfrequent cause of sudden death: it may occur at any age. In some instances, the distention of this organ appears to act by inducing apoplexy, the usual marks of that disease being found in the brain. In other cases, death appears to be due to a fatal impression analogous to shock, arising simply from the excessive mechanical distention of the organ: it is not surprising, that a suspicion of poisoning should occasionally arise under such circumstances. I have known several instances which have occurred within the last few years in this metropolis, where the individuals went to bed in their usual health after eating a hearty supper, and were found dead the following morning. On dissection, no marked changes were discovered, excepting in some cases, slight congestion of the cerebral vessels. The most striking appearance was the enormously distended state of the stomach itself.

In December 1839, a girl, aged 22, after eating a hearty supper, retired to rest. In about two hours she was found insensible, and she died in the course of a few minutes. No post-mortem examination was required by the coroner; although it is difficult to understand why, without it, there should have been any necessity for holding an inquest; as the cause of death, which was probably due to the distention of the stomach, was still left unexplained.—In April 1841, a man, aged 34, ate a very hearty breakfast, consisting of three-quarters of a pound of beef with bread, and a pint and a half of coffee. In a few minutes afterwards, he sat on a barrel to rest himself, but almost immediately fell backwards and expired.

This cause of death may be met with in persons of all ages. In November 1842, a girl, aged 13, ate a full breakfast; and about an hour afterwards she became insensible, and died in the course of a short time. The only cause which could be assigned for her death, was over-distention of the stomach with food, probably leading to apoplexy. The following case, which was the subject of an inquest in August 1841, is of a doubtful nature:—A man, aged 37, who had for some time complained of cramp in the stomach, partook of a full breakfast, consisting of coffee, beefsteaks, and fish. In about half an hour afterwards he was taken suddenly ill with violent pain and vomiting. He took some salts and cream of tartar; but became worse, and died about three hours after the meal. The verdict was, that the man "died by the visitation of God;" whereby the case was left in precisely the same obscurity as before the inquiry!

Rupture of the stomach has been observed to occur sometimes as a consequence of over-distention, combined with efforts at vomiting; although in other instances the rupture has taken place when there was but little food found in the stomach. Death is, of course, a speedy consequence of this accident: hence no difficulty can arise in practice with regard to it, because a post-mortem examination would enable the practitioner at once to determine the cause. For a fatal case of this kind, in which there was no apparent disease of the organ, see Medical Gazette, ii. 182.

Rupture of the gall-bladder and gall-ducts, as well as of the impregnated uterus or its appendages, may also suddenly give rise to alarming symptoms of a suspicious kind in a previously healthy person. Death in such cases commonly takes place from peritonitis. The rules for forming a diagnosis are similar to those already described: a post-mortem examination suffices to develope the real nature of the case.

When called to examine a case of suspected narcotic poisoning, and the symptoms have occurred soon after a meal, the practitioner must remember that although a full meal is a very common exciting cause of apoplexy, this is not the case with any simple medicine, liquid or solid, which may have been swallowed by the patient. Should the symptoms follow the taking of a draught or any kind of medicine, the circumstances becomes much more suspicious, because the occurrence of apoplexy in such a case would be a pure coincidence:—all we can say is, that it may happen—in proof of which we may refer to the case mentioned ante (page 49,) and then we require other circumstances to aid our judgment. In the case of *Sir Theodosius Boughton*, the narcotic symptoms supervened in two minutes after he had taken what was supposed to be, a simple purgative draught; and it was this fact, no doubt, that strongly influenced the jury in their verdict. The draught, it was presumed, had contained laurel-water; and with some strong facts in favour of this presumption, they were unwilling to infer that the symptoms under which the deceased laboured after taking it, were owing to a coincidental attack of apoplexy or epilepsy. In all such cases, it can never be assumed that the medicine taken was the cause of the symptoms, unless we suppose it to have been a poison; while when the symptoms follow an ordinary meal, apoplexy may be a natural result,—at least it is not absolutely necessary, in order to account for them, to suppose that the food contained any poison.

CHAPTER VI.

ON THE TREATMENT OF CASES OF POISONING—ALEXIPHARMICS—ANTIDOTES—SPECIFIC REMEDIES—CHEMICAL ANTIDOTES—THEIR MODE OF OPERATION—INSOLUBLE POISONS—GENERAL PRINCIPLES OF TREATMENT—THE STOMACH PUMP—EMETICS—ELECTRO-MAGNETISM—TABLE OF POISONS AND THEIR CHEMICAL ANTIDOTES.

The treatment of a case of poisoning must vary, not only according to the nature of the substance swallowed, but according to the period of time which has elapsed before medical assistance is demanded. Under the description of each poison a full account will be given of the various remedies which it has been proposed to employ. At the present time, therefore, it will be only necessary to state the general principles upon which the treatment of cases of poisoning is based.

ANTIDOTAL TREATMENT.—It was formerly supposed that there were substances which, if taken before or after the administration of poison, would counteract its effects. The old Mithridate was a compound of this description, made up of a variety of aromatic extracts. Its preparation was of the most complex kind, both with regard to the number of its constituents, the proportions in which they were mixed, as well as the precautions to be taken in mixing them. Hence, whenever it failed, which was always when real poison was administered, the failure was conveniently ascribed to something having been omitted in its preparation.

In later times, physicians believed in the existence of what are called in the old books *Alexipharmics* (ἀλεξω, to help or assist, and φάρμακον, medicine.) The name was applied to substances which were considered to exert a specific action on poisons; *i. e.* that they had the power, by acting on the constitution, of completely neutralizing their dangerous effects. These superstitious notions disappeared as chemical knowledge began to spread; and then a mode of treatment, founded on the *chemical* properties of poisons, was first introduced. This class of substances is now known under the name of *Antidotes* (ἀντιδοτον, remedy against poison.)

By employing the term antidote, it must not be understood that we mean a *specific* for any individual poison. The operation of antidotes is purely chemical, and their great use depends, first, upon their effects being either immediate or extremely rapid; and, secondly, on their action being local, *i. e.* on their producing an alteration in the nature of the poison by mere contact. It is obvious, that unless these substances had an *immediate* operation, they would be of no service; and we have a very easy and at the same time a simple method of testing their efficacy, by the fact that the same or similar effects ought always to be produced in a common test-glass as in the human stomach. If the alleged antidote do not chemically change the nature of the poison out of the body, it cannot be expected to have any such effect in the body, and hence it should be rejected. It is not here, as in ordinary medical practice, that the substance is given empirically; *i. e.* without our being able to explain the mode in which it operates: that mode must, in the case of an alleged antidote, be made visible and apparent, or we can place no confidence in the treatment. Whenever we depart from this principle, we lose ourselves in uncertainty; and thus it is, that substances have been frequently extolled as antidotes, and have had a certain repute for a time, but have long since passed into oblivion. Charcoal was formerly given in cases of poisoning by arsenic, and it was supposed with very good effect. Experience, however, has now shown, that the good effects under such treatment must have been really due to other causes which at the time escaped observation.

All toxicologists agree, with the few exceptions to be presently stated, that there is no substance at present known which possesses a *specific effect* in removing the symptoms produced by poison. The so-called antidotes act upon the poison, and not upon the body: their action is, therefore, strictly of a chemical nature. Some substances, it is true, are useful as stimulants, and act by rousing the nervous system. On this principle we may explain the good effects produced by a strong decoction of coffee in a case of poisoning by opium, or by ammonia, in poisoning by prussic acid. Dr. Christison considers that there are two poisons the secondary effects of which may be subdued by the administration of certain substances. These must, of course, operate by exciting in the system an action capable of neutralizing that established by the poison. The remote operation of lead may be sometimes corrected by mercury given to salivation, while the violent salivation caused by mercury, may be occasionally corrected by nauseating doses of antimony. (On Poisons, p. 41.) Mr. Allison has more recently stated, that chlorate of potash possesses the property of subduing mercurial ptyalism; and that, from the results of numerous trials, he believes this salt to stand in an antagonistic relation to mercury. (Med. Gaz. xxxviii. 983.) This comprises, I believe, all that is at present known concerning what some have denominated *constitutional* antidotes. We know of no substance that will thus subdue the effects of arsenic; but it is not improbable that, under a proper system of therapeutical inquiry, more agents of this kind, *i. e.* bodies possessing a counteracting power over this and other poisons, may be hereafter discovered.

Our inquiries respecting antidotes will therefore be limited to those substances which have a decided chemical operation, and alter the form of the poison. According to Orfila, an antidote should be a substance possessed of the following properties:—1. That it should be taken in large doses without danger; 2. That it should act upon the poison whether liquid or solid, at a temperature equal to or below that of the human body; 3. That its action should be rapid; 4. That it should be capable of combining with the poison, although enveloped in mucus, blood, or other substances contained in the stomach; and, 5. That it should entirely destroy the deleterious properties of the poison. He divides antidotes into two classes—1. Those which render the poison perfectly inert, such as the soluble sulphates in poisoning by the salts of lead or barytes, or the soluble chlorides in poisoning by nitrate of silver; and, 2. Those which, by combination, merely render the substance less poisonous, *e. g.* albumen in poisoning by the salts of mercury and copper. (Toxicologie, i. 18.) The larger number of antidotes will probably be found to act rather by diminishing, than by entirely destroying the noxious properties of the substance taken.

Mode of operation of antidotes.—This has been already stated to be of a purely chemical nature, and it now remains to be explained that the principle of their operation is, *either to render the poison less soluble, and therefore less liable to be absorbed into the body*,—or to neutralize and convert it, as in the case of a mineral acid or alkali, to a neutral or harmless chemical salt. There are numerous instances of both of these modes of operation. The greater number of chemical antidotes act merely by rendering the substance less poisonous, by diminishing its solubility in the mucous fluids of the stomach. In addition to the cases already mentioned, the use of carbonate of lime in poisoning by oxalic acid, furnishes a good illustration of this mode of operation,—the oxalate of lime, from its comparative insolubility, not acting as a poison. In rendering a poison insoluble by the administration of an antidote, it is necessary to bear in mind, that the substance used for this purpose should be itself inert, or we shall only be substituting one poison for another. I have heard of a case in which it was proposed to give subacetate of lead in poisoning by oxalic acid; and this poisonous salt has been very recently proposed by a French chemist, as an antidote to the alkaline sulphurets! In another instance, nitrate of silver was suggested as an antidote for prussic acid. Admitting that oxalate of lead, the sulphuret of lead, and cyanide of silver, are insoluble, in water,—the compounds required to form them are highly injurious, and the cyanide of silver may itself create a poisonous action on the body. The proposal of such a mode of treatment can only proceed from an erroneous theoretical view of the subject.

Some chemical antidotes do not act by rendering the poison insoluble, but by simply neutralizing it, and depriving it of its active poisonous properties. This mode of operation applies only to a limited number of substances, namely, the mineral acids and alkalies. Thus, in poisoning by either of the three mineral acids, magnesia and its carbonates are the antidotes commonly selected—a soluble salt of magnesia being formed in either case, and the corrosive properties of the acid destroyed. On the other hand, in poisoning by potash, soda, or ammonia—vinegar, lemon-juice, or a solution of citric or tartaric acid, is given, and a harmless neutral salt is formed.

It is a rule which it is almost trite to state, that as antidotes act locally, *i. e.* on the poison and not on the constitution, their efficacy entirely depends on their *timely employment*. In a large number of cases, it must be confessed, they have utterly failed, for the simple reason, that they have been administered too late. The poison has either been absorbed into the body, and thereby carried beyond the reach of any antidote ; or it has produced such extensive destruction in the stomach and other parts, as to render all treatment perfectly hopeless. This, however, must not discourage us from the use of these antidotes so long as there is life. Hundreds of persons who have been drowned cannot be restored to life : but this want of success is no reason for our refusing to apply the means for resuscitation to every future case of apparent death by drowning. In the same way we are bound to use every reasonable effort in a case of poisoning, although it may be so far advanced as to be apparently beyond the reach of all remedies.

It has just been stated, that some antidotes act beneficially by neutralizing the poison, if acid or alkaline : but we must beware of drawing the inference, that mere neutralization is all that we have to regard in the treatment. We are bound to consider whether the neutral salt formed may or may not be injurious of itself; for it is obvious that if it be injurious, we are only substituting one poison for another. Owing to false reasoning of this kind, some have adopted a very improper method of treating cases of poisoning by oxalic acid: —they have employed the alkaline carbonates, *i. e.* of potash, soda, and ammonia ; but the soluble oxalates of these alkalies are themselves poisonous, therefore the use of the alkaline carbonates should be avoided. They chemically neutralize the acid, but they do not prevent it from exerting a poisonous action.

Insoluble poisons.—Having thus considered the action of antidotes in regard to *soluble* poisons, the question naturally arises—What is to be done in those cases where the poison is just as *insoluble* as any antidote could make it? It would be wrong to infer, that because a substance is insoluble in water, it cannot act as a poison: there are some very active substances which are quite insoluble in water. Among these may be mentioned—the arsenite, carbonate, and subchloride of copper,—the carbonate of lead, carbonate of barytes, the ammonio-chloride of mercury, calomel, and yellow arsenic, or the sulphuret of arsenic. Most of these are well known to be highly poisonous, although, perhaps, they do not act so rapidly, nor in such *small* doses as their analogous soluble compounds. The admission that such insoluble substances are poisonous, may appear to be opposed to the principle on which one class of chemical antidotes has been hitherto supposed to act ; but it must be remembered, that the insoluble compounds produced by antidotes, have been found by experience to be far less active than the substances for which they are administered—they do not appear to be absorbed so rapidly, and there is, therefore, a better chance of saving life. This, of course, is a matter of experience. The sulphate and carbonate of lead, for example, are equally insoluble in water :—but observation teaches us, that the carbonate is a decided poison, while we have no reason to believe that the sulphate is possessed of poisonous properties. Hence we do not say that an alkaline sulphate acts beneficially, merely because it converts the salt of lead into an insoluble compound, for an alkaline carbonate would do this,—but because, in addition to its insolubility, there is reason to believe that the compound produced is inert. In poisoning by a soluble salt of copper, we may, by giving an alkaline carbonate, throw down the copper in the form of an insoluble carbonate ; but experience has taught us that, under these circumstances, we should only be changing one poison for another.

It was formerly the custom to give a solution of sulphuret of potassium in cases of poisoning by arsenic:—it was known that the sulphuret of arsenic was *insoluble;* hence it was rather hastily inferred that it was *inert.* We must now look upon it as a sort of miracle, that persons thus treated ever recovered. The alleged antidote itself, the sulphuret of potassium, is a poison; so that if the patient did not die from the poison originally swallowed, his life was endangered either by the sulphuret of arsenic resulting from the action of the antidote, or by the antidote itself! The inefficacy of an alkaline sulphuret as an antidote to arsenic was experimentally proved by Casimir Renault, so far back as the year 1802. He found that all of these sulphurets destroyed animals, to which small doses of arsenic had been given, in a shorter time than arsenic itself. (Nouvelles Expériences sur les Contre-Poisons de l'Arsénic, Paris, Fructidor, An. ix. p. 25.) He further proved that the numerous alleged cures by alkaline sulphurets and sulphureous waters, must have been due to other causes (ib. p. 40.) The researches of Renault are well calculated to show the inefficiency of the methods adopted by some modern experimentalists, in order to determine the antidotal properties of substances.

It has been supposed that the action of these insoluble poisons is explicable on the supposition that they may become soluble, or at least capable of suspension, in the acid and mucous fluids of the stomach. This may be, to a certain extent, true; but in one case of poisoning by carbonate of copper, the green compound was found spread over the whole of the lining membrane of the stomach and intestines. The muriatic and acetic acids in the gastric juice are in too small a quantity, to allow us to suppose that the insoluble is thereby converted into a soluble poison to any extent. Besides, the discovery of the insoluble poison in the viscera, as in the case just described, renders it probable that the acids contained in the fluids of the stomach, are not so much the cause of poisoning, as the suspension and mechanical diffusion of the substance itself through the alimentary canal. From the foregoing considerations, then, we infer that the fact of a substance being entirely insoluble in water, is no proof that it may not act as a poison on the body.

The question arises, Are we, then, without antidotes in such cases? In a case where an excessive dose of calomel or white precipitate has been given, albumen might be administered as the only resource. It would serve at least to envelope the insoluble powder, and render the ejection from the stomach more certain. In the case of the two carbonates of lead and barytes, they might be transformed into the less injurious sulphates, simply by administering sulphate of magnesia acidulated with vinegar or lemon juice. There would be no injury to the patient in trying this experiment. The alkaline sulphates alone, it is obvious, can have no action upon carbonate of lead.

Among the most formidable of the insoluble poisons is the arsenite of copper (SCHEELE'S GREEN.) I am not aware that we have any chemical antidote for this very poisonous substance, which is perfectly insoluble in water. The hydrated sesquioxide of iron has been recommended; but, from facts to be presently stated, it is doubtful whether it can be of the least utility. Admitting that the acids of the stomach may partially dissolve the poison, and that oxide of iron can displace oxide of copper from its saline combinations, the arsenite of iron formed is just as insoluble in water, and just as soluble in acids, as the arsenite of copper. No treatment is in such a case likely to be successful, except that which is directed to the mechanical expulsion of the poison from the alimentary canal.

There are some other substances for which we have no chemical antidotes: such as opium, the iodide of potassium, strychnia, cantharides, and the whole tribe of narcotico-irritant or vegetable poisons. The claims of animal charcoal, a substance which has been lately recommended as a universal antidote, will be presently considered. It has been recommended, in cases of poisoning by iodide of potassium, to give large doses of farinaceous substances. It is impossible to conjecture on what principle such substances are recommended: for they produce no decomposition of the iodide, but a mere mixture. Even the production of the blue iodide of farina in poisoning by iodine places the patient in no better position. While there is no proof whatever that a combination with starch renders iodine innoxious, there is great reason to believe that it is still capable of acting as a poison. Dr. Lawrie considers that in one case in which he administered the blue iodide of farina medicinally, it occasioned the death of the patient. (Med. Gaz. vol. xxvi. p. 589.) In recommending substances of this kind as antidotes, some appear to consider that the treatment of poisoning, consists merely in producing any sort of chemical change in the poison, forgetting that it is to the nature of the change, and not to the mere fact of its production, that we are bound to look for beneficial results. As other instances of this kind of practice, I would refer to the suggested employment of lime-water in poisoning by corrosive sublimate, whereby red oxide of mercury, itself an active poison, is formed of ammonia, as an antidote in poisoning by opium, the only effect of which is to precipitate the morphia, without in any way diminishing its poisonous properties; or of a solution of sulphate of iron in poisoning by prussic acid, which, in the absence of an alkali, can have no effect at all. The occurrence of such cases clearly proves the necessity for basing the antidotal treatment of poisoning on some well-defined principles.

As we are not acquainted with any general antidote in cases of poisoning, the treatment must be varied according to the kind of poison. It is therefore advisable that we should, if possible, obtain some previous knowledge of the nature of the substance taken. Commonly a suspicion exists;—some of the substance may be procured, or some of the matter brought off the stomach may, by its smell, colour, or chemical properties, lead to a suspicion of its nature. A diagnosis may often be obtained by observing the colour of the matter first vomited. In poisoning by sulphuric acid it is black; by nitric acid, brown, yellow, or greenish; the same in poisoning by muriatic acid; in the case of oxalic acid it is of a coffee colour, or of a dark greenish-brown. In all these cases the liquid is acid, and acts upon organic colouring matter, *e. g.* the dyes of dresses. In alkaline poisoning, there is a strong alkaline reaction in the vomited liquids. In poisoning by the salts of copper, the matter vomited is of a greenish or blueish colour. Some substances are at once denoted by the odour; as prussic acid, oil of bitter almonds, ammonia, opium and alcohol. In other instances, the poison can be detected only by chemical processes, and for the ready application of tests, an extensive acquaintance with practical toxicology is required. When none of the substance can be procured, and no information respecting it can be obtained, we may in some instances be guided, first, by the nature of the symptoms, and, secondly, the time at which they commenced after the substance was swallowed.

It has been already stated, that *irritant poisons* are known, from whatever kingdom they may be derived, by their occasioning speedily severe pain in the abdomen, with violent vomiting and purging. Similar symptoms may, as we have seen, proceed from certain diseases; but if there should be any moral ground to suspect poison, we are bound to employ the means resorted to in cases of poisoning. These means are: first, *the removal of the poison from the stomach;* and, secondly, *the employment of chemical antidotes.* The removal of the poison from the stomach, may be effected either by emetics or the stomach-pump. This is the great point to which we must look for the safety of the patient. All other treatment, even that of antidotes, must be subordinate to this. So long as any portion of the poison remains in the stomach, the patient is not safe; and if we delay the employment of means to remove it, he will die, although the stomach may be subsequently entirely cleared of the poison. Cases of this description have repeatedly occurred at Guy's Hospital, the patient having been brought in when too late. It has sometimes happened that the party first called to see the patient, has been too much occupied in seeking for or administering antidotes, and has neglected to empty the stomach, which is the first and by far the most important consideration in practice. The two kinds of treatment may go on together, *i. e.* we may promote vomiting by the administration of water containing the antidote.

Emetics.—With regard to clearing the stomach, the irritant poisons in general act as powerful emetics, and therefore all that we have to do is to promote vomiting by the free administration of warm water. In the case of oxalic acid, however, it is desirable to avoid giving much water, as the poison is dissolved and rapidly diffused over a large surface of the intestines, whereby it becomes more easily absorbed. If the vomiting should cease, or if it should not have taken place as an effect of the poison, we must then give an emetic of sulphate of zinc or copper, *i. e.* from fifteen to twenty grains at a dose, and unless attended with marked effect, this must be repeated every quarter of an hour. Ipecacuanha and tartar emetic should be avoided, for they are liable to produce excessive nausea, and during this state the poison becomes more rapidly absorbed. Supposing that we have not at hand the sulphate of zinc or copper, there is scarcely a house in the country that does not furnish an emetic in the shape of common mustard. From a teaspoonful to a dessert-spoonful of this, in half a glass of warm water, may be given to the patient every five or ten minutes, according to the effect. The back of the throat may be at the same time tickled with a feather. If there should be no mustard, then a thick mixture of yellow soap and warm water may serve as a substitute.

It is right to bear in mind, that most of these emetic substances are themselves irritant, and therefore a proper degree of caution must be observed in giving them. In all cases where the symptoms are those of irritant poisoning, the administration of viscid or mucilaginous liquids is advisable. Barley water, decoction of linseed, flour and water mixed in the form of a paste, albumen, or a mixture of oil and lime water, or yellow soap and water may be freely given. These viscid substances serve to protect the coats of the stomach, to lock up the undissolved particles of poison, and thereby favour its expulsion during the act of vomiting.

A second method of clearing the stomach is by the use of the *stomach-pump.* This most useful instrument has been undoubtedly the means of saving life in many cases. On some occasions it may supersede the necessity for resorting to emetics; at others, it will effectually clear the stomach, where emetics will not answer; and lastly, it admits of the introduction of the antidote and the speedy removal of the compound formed. In a case where nearly an ounce of arsenic had been swallowed, I found the stomach so cleared by the active use of this instrument, that after death not the fiftieth part of a grain of the poison could be detected in the organ. The fact of the patient having died, may appear to show that in this instance the instrument was of little service; but the female was only brought to the hospital many hours after the poison had been taken. The result, nevertheless, clearly proves how perfectly this instrument acts, even with regard to a comparatively insoluble poison like arsenic. The employment of the stomach-pump requires great care: serious accidents have more than once arisen, even in the hands of those who have made anatomy a branch of their studies. In one instance it is reported, that after death some of the injected liquid was found in the lungs, into which organs it had been thrown instead of the stomach. Should any violence be used, the end of the tube, if of ivory, may tear and lacerate the membranes covering the throat and gullet, or even the stomach itself, of which many instances have occurred. There are several varieties of the stomach-pump. That is the best which is the most simple, and requires the least shifting of its parts. One has been lately introduced, in which the action of the instrument is reversed simply by moving the syringe backwards and forwards, without any unscrewing. In children, the use of a common catheter has been recommended, to which a syringe may be fastened by a piece of wet skin or bladder.

There are some cases in which it is advisable not to employ the stomach-pump, but to trust to vomiting. I allude to those instances in which the poison taken, has been a mineral acid or caustic alkali, bromine, iodine, or in short a corrosive substance of any kind whatever. In poisoning by any of the corrosives, those parts of the body which come in contact with the substance, are chemically acted on and destroyed. The attempt to pass an instrument in such a case, might lead to the laceration of the softened membranes, and in some instances to the perforation of the œsophagus or stomach. Such cases may be easily distinguished from others, for on opening the mouth the chemical action of the poison may be plainly seen on the teeth, gums, tongue, and the back of the throat.

The Narcotic and Narcotico-Irritant Poisons are known by the peculiar cerebral symptoms which they produce. Our treatment must be directed to the removal of the poison from the stomach; and there is no instance in which the stomach-pump has been so successfully employed as in this form of poisoning, when the poison has been taken in solution. If the instrument be not at hand, then we must employ one of the emetics already described; but it is proper to remember, that emetics never act readily unless the person is kept roused. On no account should we allow the individual to give way to the heavy feeling of sleep which commonly oppresses him. There is no plan so well adapted for recovering a patient, or so successful in its results, as the passing of shocks along the course of the spine and in the cardiac region by means of an electro-magnetic apparatus. Mr. Sibson, of the General-Hospital, Nottingham, has applied this method of treatment most successfully, in poisoning by opium, when every other plan had failed. Mr. Tubbs, of Upwell Isle, informs me, that, by the application of gentle shocks from a battery of this kind, he succeeded in saving the life of an infant only three weeks old, which had been poisoned by an over-dose of Godfrey's cordial. In the absence of this apparatus, the patient may be kept roused by causing two active persons to walk about with him,—by dashing cold water upon the chest, back, and head,—by rubbing the chest and backs and palms of the hands with compound camphor liniment, or any other stimulating embrocation. It has been recommended to employ flagellation to the palms of the hands and soles of the feet. In the cases of young infants, it has been found sometimes beneficial to plunge them into a warm bath, and suddenly raise them into the cold air; this is said to have acted as a very effectual stimulant. Ammonia may be cautiously used to the nostrils in cases of poisoning by opium; but we must remember that it is a powerful stimulant; and if the strong vapour be allowed to be too fully respired, it may produce serious after-consequences. When the poison has been removed, and the power of deglutition exists, a very strong decoction of coffee may be frequently given; or, should the individual be unable to swallow, this may be introduced by the stomach-pump.

There are no chemical antidotes for these poisons; *i. e.* there is no substance which will so decompose them in the stomach as to prevent their absorption and diffusion. Our treatment, therefore, is confined to the expulsion of the poison, and as far as possible to the counteraction of the symptoms by ordinary therapeutical means; not to the production of any chemical change in the poison itself. Infusion of cinchona or oak bark may precipitate the alkaloid, but it is questionable whether the patient will derive any benefit from the administration of liquids containing tannin. Vinegar was formerly used and recommended as an antidote for opium, but we now know that it tends to render the poisonous part of the drug more soluble, and it thus aggravates the mischief. Ammonia has been also recommended, but it can only act beneficially as a stimulant after the removal of the poison.

The narcotico-irritant poisons are commonly taken in substance, as under the form of roots, leaves, berries, or seeds of the respective vegetables. Emetics, purgatives, and laxative enemata, constitute the treatment which should be pursued, The removal of the poison, even after it had been some time in the stomach, has been often attended with the best effects. If these substances have been taken in the form of decoction or infusion, the stomach-pump may be beneficially employed.

TABLE OF POISONS AND ANTIDOTES.

	POISONS.	ANTIDOTES.
	Non-Metallic Poisons.	
Mineral acids	Sulphuric Sulph. Indigo Nitric Muriatic Nitro-Muriatic Nitro-sulphuric	Magnesia, mixed with milk or water — Carbonate of lime — Compound chalk powder — Mixture of soda — Carbonate of soda diluted.
Vegetable acids	Oxalic Tartaric	Carbonate of lime.
Salts	Binoxalate of potash	Carb. lime — Sulph. lime, dissolved in water.
	Bitartrate of potash	Carbonate of soda diluted.
Alkalies	Potash, soda, ammonia, and their carbonates	Vinegar and water, equal parts — lemon-juice—citric acid — oil.
Salts	Baryta and its soluble salts	Sulphate of soda, magnesia, potash, or lime.
	Carbonate of baryta	A mixture of sulphate of magnesia and vinegar, diluted.
	Alum	Carbonate of soda, or sesqui-carbonate of ammonia.
	Metallic Poisons.	
	Arsenic and soluble Arsenites—Arsenic acid	A mixture of oil and lime water—Hydrated-magnesia? — Hydrated sesqui-oxide of iron?
	Corrosive sublimate and salts of mercury	Albumen diffused in water—Gluten or flour diffused in water.
	Soluble salts of lead	The alkaline sulphates.
	Carbonate of lead	Sulphate of magnesia and vinegar diluted
	Soluble salts of copper	Albumen—Milk—Gluten—Flour and water.
	Tartarized antimony	Decoction of oak bark, or cinchona —Tincture of kino, or catechu— Magnesia.

Chloride of antimony	Carbonate of soda—Magnesia.
Salts of tin	Milk—Carbonate of soda—Magnesia.
Sulphate or acetate of zinc	Milk—Carbonate of soda—Magnesia.
Sulphate of iron	Carbonate of soda, or sesqui-carbonate of ammonia.
Nitrate of silver	Chloride of sodium.

Narcotic Poisons.

Opium—Hyoscyamus	Emetics—Stomach-pump—Cold affusion—Strong decoction of coffee—Electro-magnetism.
Prussic acid—Essential oil of almonds	Cold affusion—Mixed oxides of iron in water.

The special details of treatment will be given hereafter, in speaking of the poisons individually.

From the remarks already made, it will be observed, that most of the substances in the column of antidotes act only by diminishing the solubility of the poison, and therefore by rendering it less noxious.

CHAPTER VII.

TREATMENT OF POISONING CONTINUED—GENERAL ANTIDOTES—CHARCOAL—ITS INEFFICACY—ANTIDOTES TO THE MINNRAL ACIDS—ANTIDOTES TO ARSENIC—HYDRATED OXIDE OF IRON—HYDRATE OF MAGNESIA—EXPERIMENTS—ANTIDOTES TO CORROSIVE SUBLIMATE—ALBUMEN—CHLORIDE OF TIN—SOURCES OF FALLACY IN THE USE OF ALLEGED ANTIDOTES—GENERAL TREATMENT OF CASES OF POISONING.

MANY attempts have been made of late years to discover some general antidote which might be administered in every case of poisoning; but these have dened in failure. Nothing but the complete expulsion of the substance from the body can give security to the patient, or hope of success to the medical attendant.

Charcoal powder was recommended as an antidote to arsenic, by M. Bertrand, nearly forty years ago; but, from experiments made by Orfila and others, it appeared to act only by mechanically enveloping the poison. M. Bertrand is reported to have swallowed with impunity five grains of arsenic in one dose, mixed into an emulsion with charcoal: but as this is not the way in which arsenic is swallowed as a poison, the fact establishes nothing in a practical point of view. Dr. Christison states that charcoal has been proved to be destitute of all efficacy, when not administered until after the arsenic is swallowed; and this is undoubtedly the truth. If M. Bertrand had mixed the arsenic with fine clay or sand, he would probably have found these substances equally efficacious! The fact is, large masses of insoluble powders tend to block up the orifices of the absorbents, and prevent the absorption of the poison: but it is quite a misapplication of language to call such substances *antidotes*. (See Flandin, Traité des Poisons, i. 587.) The use of purified *animal charcoal* has been lately proposed by Dr. Garrod as a general antidote in cases of poisoning, but especially for those noxious substances which belong to the organic kingdom. He states that he found this kind of charcoal to form compounds with arsenious acid and other mineral substances, removing them from their solutions, and that it is quite equal, if not superior, to the hydrated oxide of iron as an antidote to arsenious acid; that the compounds which animal charcoal forms with poisonous principles, have no injurious action on the body, and the antidote itself may be given to almost any amount, as it is perfectly inert. (Pharm. Journ. Jan. 1, 1846, p. 325.) The quantity required for neutralizing (?) strychnia amounted, according to him, to about 240 times the weight of the poison; but the proportion required for the separation of arsenic or corrosive sublimate is not stated. Most of Dr. Garrod's experiments appear to have been performed by administering to the animal, the poison *previously well mixed with charcoal.* The results, therefore, like those obtained by Bertrand in 1813, do not appear applicable to practice. The charcoal probably acted in these cases mechanically, especially as it was only found efficacious when given in very large bulk; and it is not improbable that an equal quantity of pipe-clay would have had a similar effect. Dr. Garrod says, that "in the experiments with the pure alkaloids, the antidote was given with the poison: in the case of the more mild vegetable poisons the antidote was administered ten or fifteen minutes afterwards, and mostly with a favourable result. (Pharm. Journal, April 1846, p. 441.) These experiments do not appear to me to be sufficient to justify us in ranking animal charcoal as an antidote. This substance has been long known to possess the power of removing colours and odours from organic liquids; and in a work published in 1844, (Manual of Med. Jur. 1st ed. p. 248) I stated that this was an objection to the use of animal charcoal in discolorizing a suspected solution of opium; but, with the exception of the instances referred to by Dr. Garrod, I am not aware that animal charcoal possesses the property of precipitating or combining with any mineral poison to form an insoluble inert compound. The following experiments appear to me to be decidedly adverse to the view that it can operate as an antidote to arsenic under any circumstances whatever. A grain of arsenious acid was dissolved in five drachms of water, and well shaken with sixty grains of animal charcoal, for a quarter of an hour. The liquid was filtered and tested, when arsenic was found in it by all the tests, just as abundantly as if no animal charcoal had been employed. Whether any minute particle of the poison had become locked up in the mass of charcoal left on the filter, was not so material, as the fact that a very large quantity of arsenic was still freely dissolved in the filtered liquid. In this case the poison was perfectly dissolved, and therefore in a state best fitted for this alleged mode of separation. In the next experiment finely-powdered arsenic was employed. One grain was well mixed in five drachms of water, with sixty grains of animal charcoal, and shaken for a quarter of an hour. On allowing the tube to stand at rest, the white grains of arsenious acid were observed to be lying beneath the charcoal at the bottom of the tube; there had been evidently no change, a result for which, considering the great insolubility of arsenious acid, one might have been prepared. The mixture was then boiled for twenty minutes, the loss by evaporation being made up: the filtered liquid was found, after half an hour, to be as strongly impregnated with arsenic as if no charcoal had been present. In the next experiment one grain of corrosive sublimate was dissolved in five drachms of water, and sixty grains of animal charcoal added. The mixture was shaken for a quarter of an hour, and filtered. The usual tests for corrosive sublimate showed its presence in abundance in the filtered liquid. It therefore appears to me, that animal charcoal, even when used in *sixty times* the weight of the poison, and under the most favourable conditions of perfect solution, of immediate mixture, and, in one instance, at a boiling temperature, can have no counteracting influence whatever on the poisonous effects of arsenic and corrosive sublimate. (See also Devergie, Méd. Légale, ii. 471.) If half an ounce of either of these poisons had been swallowed, it is therefore obvious that even *thirty ounces* of animal charcoal would have had no effect! With respect to the action of animal charcoal on organic poisons, I have observed that there has been an apparent removal of meconate of morphia in the case of opium, but to an extent quite insufficient to place the patient in safety. In regard to the pure alkaloids, the satisfactory results have only been obtained by Dr. Garrod with enormous quantities of charcoal, and under circumstances in which we are never called upon to treat a case of poisoning, *i. e.* where the poison has been taken *mixed* with the antidote. I therefore most fully agree with the statement made many years since by M. Devergie, that not the slightest confidence can be placed in animal charcoal as an antidote in any case of poisoning. (Médecine Légale, ii. 470, 1 Ed.) M. Bussy, who has lately experimented on this subject, has come to the conclusion that purified animal charcoal has no power of counteracting the effects of arsenic. (Gaz. Méd. May 23, 1846.)

Objections might be taken to many of the substances contained in the list of antidotes: for the efficacy of some of them in neutralizing the effects of the poison is very questionable.

Antidotes to the Mineral Acids.—Magnesia is commonly recommended, but its bulk and great insolubility render it inconvenient for administration. I ascertained that in one case of poisoning by sulphuric acid, in which magnesia had been given soon after the accident, the matter vomited, after the lapse of from half an hour to an hour, was still intensely acid and corrosive, the poison having become only very partially neutralized. I am therefore inclined to think that the frequent administration of a diluted solution of carbonate of soda or potash, would be much more efficacious than the use of magnesia or its carbonates. In poisoning by oxalic acid, these soluble carbonates, for reasons already stated (page 70,) would be inadmissible.

Antidotes to arsenic.—There are two substances which have been recently strongly recommended as antidotes to arsenic in all its forms: I allude to the hydrated sesquioxide of iron and hydrate of magnesia. It is said that arsenic (arsenious acid) forms an insoluble precipitate with both of these substances, and that the poison is rendered comparatively inert. I have placed these substances in the list of antidotes, in deference to the views entertained by some eminent toxicologists: but I do not consider it the less necessary to state here those circumstances which induce me to believe that no reliance can be placed on either of them in cases of poisoning by arsenic.

Hydrated sesquioxide of iron.—This substance is prepared by precipitating persulphate of iron by ammonia, and washing the precipitate: it is used in the moist or hydrated state. It is supposed by some to act by combining with the arsenious acid to form an insoluble arsenite of iron; but it has been clearly proved by the experiments of Taddei and Orfila, that the arsenite of iron, like the arsenite of copper, is a poison; and those who advocate its use, admit that a much larger quantity of oxide than is necessary to form the chemical arsenite, is required for any antidotal effect to follow. Dr. Brett found in his experiments, and I have ascertained the same point, that when even eight or ten parts of the dydrated oxide were mixed with one of the poison in a perfect state of solution, the arsenic was not entirely thrown down; but might still be detected in the liquid by the usual tests. Dr. Maclagan of Edinburgh states, that it requires twelve parts of oxide as a hydrate, and sixty parts when dried, to neutralize one of arsenic; therefore something more is necessary than the formation of an insoluble arsenite of iron, admitting that this compound is really produced. According to this view, if an ounce of arsenic has been swallowed, and none of the poison ejected, twelve ounces at least of the hydrated oxide should be given immediately, in order to produce any good effects; and this is on the assumption that the poison is in a state of perfect solution in water! But as arsenic is almost always taken in the form of *powder*, and is very little soluble in water, it appears to me that all experiments performed with the hydrated oxide of iron on a boiled and filtered solution of the poison, have not the least practical bearing on the question of treatment. Devergie states that oxide of iron, unless taken in a dose equivalent to *thirty-two times* the weight of the poison does not prevent its action as such. Thus, if a person has taken three or four drachms of Arsenic—a common dose—at least a pound of the oxide must be given to have any antidotal effect! (Médecine Légale, ii. 475.)

In order to ascertain whether this substance could really be considered a *chemical* antidote under the circumstances in which arsenic is commonly taken, I mixed a quantity of the hydrated sesquioxide of iron, obtained by precipitating with ammonia six ounces of a strongly saturated solution of the persulphate of iron, with forty grains of finely powdered arsenious acid, adding about two ounces of a mixture of albumen and water, as a substitute for the mucus of the stomach, and making the whole quantity up to sixteen ounces with distilled water. The mixture was well agitated, and kept at a temperature of about 75° for a week, at the end of which period the greater part of the arsenic still remained undissolved at the bottom of the vessel. As this poison is frequently taken in coarse lumps, and almost always in very large quantities, in the state of powder, it is not possible to conceive, under these circumstances, that oxide of iron should exert any chemical action upon it of an antidotal nature.

In order to test further, the alleged value of the oxide of iron as a chemical antidote, the following experiment was performed. Sixty grains of finely powdered arsenic were mixed with the hydrated oxide of iron, recently precipitated from eight ounces of a saturated solution of persulphate by ammonia, —a sufficient quantity of this alkali being left to produce a strong reaction on test paper. The whole quantity of water was made up to twenty fluid ounces. This mixture was frequently agitated, and kept for six hours at a temperature of 98° in a water-bath. It was allowed to remain for a fortnight, being occasionally shaken during that time. On examination it was found, that a large portion of the powdered arsenic still remained undissolved, and uncombined with the oxide of iron at the bottom of the vessel. On drying a portion of this sediment on plate glass, the arsenious acid was easily detached from the oxide of iron. No portion of arsenic was held dissolved in the liquid, which was no longer alkaline. Reinsch's test, however, showed that some part of the arsenic was either mechanically mixed or chemically combined with the oxide; and there is no doubt that so much as had been dissolved by the water and the alkali, had become precipitated. The quantity of oxide of iron here used was very great.

It has been stated that an insoluble combination of arsenic with oxide of iron is not formed unless an excess of ammonia is present; and Dr. Christison considers that a triple compound is formed of arsenious acid, ammonia, and a large excess of oxide of iron. Orfila (i. 365) found that arsenite of iron is itself a poison, only less active than arsenious acid from its being less soluble: —he inferred from his experiments that this compound of arsenic and sesquioxide of iron acted as a poison to animals, owing to its being slightly soluble in the gastric juice. By giving a larger quantity of oxide, its insolubility was increased, and then, he states, it possessed antidotal powers. But still it appears to me, that this explanation does not meet the question. What is to render the powdered or coarse arsenic soluble, in order that the insoluble oxide may combine with it? Among those who have found that it is entirely destitute of antidotal powers is Dr. Cramer. He administered arsenic to ten rabbits, and employed the oxide of iron as a counter-poison; but without any good results. (Schneider's Ann. 1836, i. 455.) From many experiments which I have performed in order to determine the value of this alleged antidote in a chemical point of view, I have obtained the following results:—1. When the arsenious acid is perfectly dissolved in water, and mixed and agitated with twelve or fifteen times its weight of the hydrated oxide, the poison is precipitated with it in a very insoluble form, and on drying is not separable as arsenious acid from the oxide of iron by the heat of a spirit-lamp. 2. When the poison is mixed and agitated in the state of *powder* with the oxide, there is little or no effect. The arsenic becomes mechanically diffused through the oxide, and is readily obtained by volatilization from the dried powder, in which the grains of arsenious acid are easily distinguishable. No more is precitated than cold water will dissolve, *i. e.* about one-five-hundredth part. 3. When the poison in powder is mixed with the oxide of iron rendered alkaline by ammonia, so much appears to combine with the iron, as the quantity of alkali will render soluble in cold water. The rest is diffused in granules through the oxide.

Most of the experiments in favour of this antidote, have been performed on clear and filtered *solutions* of arsenic; and therefore the results are perfectly irrelevant; since arsenic is in almost all cases taken in *powder*, and often in very coarse powder. It is said that the oxide of iron may act in the stomach by preventing the absorption of the arsenious acid as it is dissolved; but it is not easy to perceive how this can happen with sufficient rapidity to render the oxide efficacious as an antidote. Let us imagine that the powdered arsenic is mixed up into a paste, and spread over a wounded or an ulcerated surface or a cancerous breast. The absorbing surface might be covered with any quantity of hydrated oxide of iron, with or without an alkali. Is it possible to suppose that the addition of the oxide of iron will prevent the absorption of the poison wherever it is in contact with the ulcerated surface? These cases would allow the efficacy of the alleged antidote to be fairly tested; for neither vomiting nor the use of the stomach-pump could here interfere with our judgment. In introducing the oxide of iron into the stomach after the poison has been swallowed, we can do no more than lay it over the arsenic, which commonly adheres very closely to, and is often firmly imbedded in, the mucous membrane. A friend informs me that he lately examined the stomach of a person who died from poison; and he found the arsenic, mechanically mixed with the hydrated peroxide, adhering to the inflamed patches of the mucous membrane. This is exactly what might be expected from the known properties of these two substances in a solid state.

If arsenic were swallowed in the state of a filtered aqueous solution, the oxide of iron might *pro tanto* combine with it; but then its antidotal effects are so imperfect, that unless administered instantly in a very large proportion, it could be of no benefit; for it is obvious that in such a state of solution, arsenic would be speedily absorbed, and act with very great rapidity. Numerous recoveries are said to have occurred under the use of this alleged remedy, but so far as I have been able to ascertain, in severe cases, emetics and the stomach-pump were also freely used; and in the lighter cases, recovery would probably have equally taken place without it. Recoveries were said to take place formerly, under the use of the alkaline sulphurets, or albumen and milk. Flandin quotes a case in which an aged person recovered from the effects of a dose of arsenic by the use of milk with yolk of egg, and the employment of emollient enemata —oil and carbonate of potash. (Traité des Poisons, i. 340.) In the case of the *Turners* (1815,) five persons recovered from the effects of arsenic, under treatment, which we should now look upon as highly injurious. (Marshall on Arsenic, 106.) It appears probable that in this, as in other cases of poisoning, too much importance has been attached to the effect of the supposed antidote, and too little assigned to the efforts of nature and the simultaneous employment of emetics and the stomach-pump.

These objections to the oxide of iron as an antidote in arsenical poisoning are entirely based on *chemical* grounds. This substance does not fulfil any of the indications, which are clearly proved to be necessary with respect to other chemical antidotes. It will not combine with the poison in the only form in which it is taken: and when it does combine with it, *i. e.* in a form in which it is never taken, it constitutes a poisonous compound soluble in the juices of the stomach. If it really has done good in cases of arsenical poisoning, it must have been either by acting mechanically, or by producing some unknown specific effect, and its operation must be widely different from that of all other antidotes.

Still more recently, the acetate of the sesquioxide of iron has been recommended by Dr. Duflos. In experimenting with this compound, I have found that in respect to arsenic in *powder*, it is as inefficacious as the hydrated oxide, even when an alkali is added to produce effectual precipitation; and that with regard to a *solution* of arsenious acid, the poison is more readily precipitated by the hydrated oxide, than by the acetate of iron.

Hydrate of magnesia.—Magnesia and its carbonate were formerly recommended as antidotes in poisoning by arsenic: and the administration of large doses of magnesia was said to be attended with good effects. The employment of magnesia has been lately revived by a French chemist, M. Bussy, who states, that when it is but slightly calcined, it easily absorbs arsenious acid from its solution, forming an arsenite of magnesia insoluble in boiling water—an action which is especially manifested when the magnesia is in a gelatinous or hydrated state. (Gaz. Médicale, May 23, 1846.) This antidote, according to M. Bussy, perfectly neutralizes the poison; and in his opinion it is superior to all others. Some experiments that I have performed with the best calcined magnesia of the shops, which was exceedingly light and flocculent, have led me to infer that the chemical action between it and arsenious acid is so small, as scarcely to justify the application of the term antidote to it. 1. A grain and a half of arsenious acid was dissolved in water, and digested with thirty grains of magnesia diffused in water. The mixture was well shaken for a quarter of an hour and filtered. The solution was feebly alkaline. On boiling, it became turbid, a white compound being deposited (subarsenite of magnesia?) The filtered alkaline liquid contained arsenite of magnesia dissolved; and arsenic was most abundantly detected in it by all the usual tests. 2. One grain of arsenious acid dissolved in water, was digested in a similar way with sixty grains of magnesia, for a quarter of an hour. At the end of this time, a portion of the liquid filtered was examined for arsenic, and it was found to be present in large quantity, probably as arsenite. After the lapse of forty-eight hours, another portion was filtered and tested; but no arsenic or arsenite of magnesia was discovered in the filtered liquid. On examining a portion of the magma of magnesia on the filter, by dissolving it in diluted muriatic acid, arsenic was found to be freely contained in it, apparently in an insoluble form. 3. Five grains of arsenious acid were dissolved in one ounce of water, and shaken up with sixty grains of magnesia diffused in another ounce of water. Portions of the liquid were filtered and examined at the end of half an hour, one hour, two hours, twenty-one hours and forty-four hours. Arsenic was detected in each case in the filtered liquid, both by Reinsch's and the other tests for arsenic. After forty-four hours' digestion, the filtered liquid was clear, strongly alkaline, precipitable by ammonia and lime water, and copiously by a mixture of ammonia and phosphate of ammonia. It became quite milky on boiling. When boiled with a few drops of muriatic acid, and metallic copper was added, this metal acquired the tarnish indicative of arsenic. Hydrosulphuret of ammonia and diluted muriatic acid gave the usual precipitate indicative of sesquisulphuret of arsenic. The magma was not examined, since these results showed clearly that the arsenic after so long a digestion, had not been precipitated in an insoluble form, but had become a soluble arsenite. 4. In this experiment five grains of arsenious acid in powder were mixed with sixty grains of arsenic, well shaken, and allowed to digest in a warm place for forty-four hours. A small portion of this milky liquid was poured off, and the magnesia dissolved by very diluted muriatic acid. A sediment was speedily deposited, consisting of small white grains, which, on examination was proved to be arsenious acid. It is quite obvious that in the pulverulent state, there had been no chemical action between the magnesia and arsenious acid; for neither a soluble nor an insoluble arsenite had been formed. In these different experiments, it will be perceived that arsenic in *solution* was mixed with twelve, twenty, and sixty times its weight of magnesia, for periods varying from a quarter of an hour to forty-eight hours; but the arsenic was only completely separated in an insoluble form, after forty-eight hours (in No. 2,) while the magnesia was in a proportion of sixty parts; and the inferences I would therefore draw are, that ordinary calcined magnesia has only a very feeble action on arsenic when *dissolved*, and no action whatever upon that substance when in the state of solid arsenious acid:—that in its alleged antidotal operation on the solution, a very soluble arsenite of magnesia is produced, which is itself poisonous;—and that unless given in twenty or thirty times the weight of the arsenic, and allowed to remain in contact some hours, it would have no appreciable effect in diminishing the virulence of the poison. On arsenic in the state of powder, i. e. as it is almost universally taken, magnesia has no effect whatever as an antidote; and if it act beneficially, it must be by enveloping the particles of the poison, and mechanically preventing their contact with the mucous membrane of the stomach.

From Dr. Christison's experiments, it would appear that the effect depends greatly on the qualities of the magnesia. The dense (?) magnesia of the shops he found to exert but very little action in removing arsenic from its solution in water;—that a very light magnesia (largely manufactured at Belfast,) free from carbonic acid, removed about a *twenty-fifth* part of its weight of arsenic from the solution in water, when agitated with it for a few minutes, so that the presence of arsenic was not indicated by ammonio-nitrate of silver;—that the same magnesia removed about a twelfth of its weight of arsenic when agitated occasionally for a period of about eight or twelve hours;—that this proportion was removed entirely in less than three minutes when the mixture of magnesia and water had been previously heated to nearly 212°;—and that the same proportion is removed, with as much speed, at ordinary temperatures, when the magnesia was used in the form of gelatinous pulp, as thrown down in a cold solution of sulphate of magnesia, by solution of caustic potash, and washed with cold water. When this gelatinous hydrate cannot be easily procured, the light calcined magnesia he considers may be used in the proportion of between thirty and fifty parts to one of arsenic. (Monthly Journal of Med. Science, August 1846, p. 158.) These experiments refer to arsenic only in a state of *solution;* and the beneficial results appears to exist only to a very limited extent. In order that the alleged antidote should act, the poison must be dissolved: common magnesia will have no effect, but a peculiar preparation must be used, which will not be efficacious, unless employed in the proportion of about *thirty* times the weight of the poison, *i. e.* with half an ounce of arsenic dissolved, at least fifteen ounces of magnesia must be used! If the arsenic be taken, as it usually is, in the form of *powder*, there is no reason to suppose, so far as these experiments are concerned, that the magnesia will act as a chemical antidote. I have ascertained that the arsenite of magnesia formed is quite as soluble in diluted muriatic acid as arsenite of copper. The acids of the gastric secretion might therefore suffice to dissolve it; and thus bring a soluble poisonous compound in contact with the mucous membrane of the stomach. From this circumstance, and from the enormous bulk in which it is necessary to give magnesia in order to render it efficacious, I am inclined to agree with the opinion expressed by Dr. Christison in the last edition of his work, that, like charcoal, it only acts, if of any use at all, by covering the arsenical particles with its fine insoluble powder, and so preventing them mechanically from coming in contact with the surface of the stomach. (On Poisons, 362.) The gelatinous magnesia is troublesome to prepare, and requires time for the separation of the potash which is used in its precipitation.

Corrosive Sublimate—Various antidotes have been suggested for corrosive sublimate, and among these that which holds the first place is *albumen*. This remedy was recommended many years since by Orfila, and there are several instances of its efficacy on record. It is alleged to have had good effects, even when it was not taken until some time after the poison had been swallowed. A man, aged sixty-eight, swallowed a drachm of corrosive sublimate. The usual symptoms appear to have followed, but he was not seen until about three quarters of an hour had elapsed. His countenance was pale and anxious, his extremities cold, and the pulse small, hard and frequent. Emetics were given to him, with the whites of six eggs, and after vomiting violently, he recovered in three days. There was reason to suppose, however, that the effect of the antidote was aided by the application of other remedial means. On adding albumen to a solution of corrosive sublimate, a dense white precipitate is thrown down, concerning the real nature of which there are many different chemical opinions; but the only practical point which immediately concerns us is, that the poison from being soluble, is brought to an insoluble condition, and it is therefore far less likely to do mischief, than the original solution of corrosive sublimate. If the corrosive sublimate be swallowed in powder or lump, albumen will have the same effect, but it will of course operate more slowly, and less effectually. The common practice in using albumen is, to give only the white of egg; but, chemically speaking, the yolk, which is composed of the same principle, with a small quantity of yellow oil, is just as efficacious. Orfila has collected this precipitate, and administered it to animals, in order to observe whether it had any injurious effects. In three instances, he gave to animals (dogs and rabbits) *fifty grains* of the compound: in one case, as a dry powder, in the other two cases, as a jelly, washed free from corrosive sublimate. In only one instance was there slight vomiting, and all the three animals recovered; although he believes that had the corrosive sublimate been given separately from the albumen, it would have destroyed them. Other experiments which he made were not attended with such successful results. Nevertheless, he concludes that the compound may be taken in a large dose without danger; that it becomes poisonous when dissolved in albumen, but is not then so active as corrosive sublimate. (Toxicologie, i. 543.) M. Devergie expresses some doubt concerning the alleged efficacy of this antidote, and he thinks that the albumen of at least one egg would be required for the neutralization of one grain of the poison. (Médecine Légale, i. 476.) The white compound formed on mixing albumen with corrosive sublimate, is perfectly soluble in water; but it is found to be soluble in, or rather easily miscible with, a large quantity of albumen. It has therefore been supposed that too much of the antidote might be given, and the poisonous action restored. It is doubtful, however, whether albumen has any other effect upon it, than that of mechanically suspending it. Much discussion has arisen among toxicologists respecting the nature of the compound formed by albumen, when exhibited in cases of poisoning by corrosive sublimate. The great practical question is, as to how far it is capable of disarming the poison of its virulence, and upon this most are agreed, namely, that it is a useful counter-agent.

With regard to the compound formed, Orfila's opinion was, that the corrosive sublimate was reduced to the state of calomel by albumen, and thereby rendered inert. Lassaigne stated, from his experiments, that the albumen combined directly with the corrosive sublimate, and formed an insoluble substance. A writer in the Dublin Journal of Medical Science (May 1844,) has lately called the attention of toxicologists to the experiments of Professor Rose, which correspond in their results with those performed by himself. Prof. Rose considers the compound to consist of albumen united to the peroxide of mercury; and there is no doubt that a compound similar to, if not identical with it, may be at once formed by rubbing up fresh albumen with hydrated peroxide of mercury. The same may be procured by precipitating with albumen "a solution of pure pernitrate of mercury, as nearly neutral as possible." If albumen be added to the protonitrate of mercury, the protoxide is thrown down of a grayish-black colour. In performing lately some experiments on the subject, I have found that the compound, produced directly by the admixture of albumen with the hydrated peroxide of mercury, possesses all the chemical properties of that produced by the action of albumen on corrosive sublimate. Thus, it underwent similar changes when treated with chloride of tin, metallic copper, caustic potash and concentrated muriatic acid; but there was one difference, namely, that a small portion of corrosive sublimate was held combined with the precipitate formed in a solution of that poison by the addition of albumen. Albumen was added to a solution of corrosive sublimate, in sufficient quantity to produce the usual dense white precipitate, but not to redissolve it. The clear liquid was poured off, and the precipitate was afterwards thoroughly washed on a filter, until the washings gave no indication of the presence of corrosive sublimate. On adding potash to a portion of the precipitate, there was no apparent change, but on boiling a larger quantity of it in water, filtering and evaporating on a glass plate to crystallization, some minute white prismatic crystals were obtained, which were immediately turned scarlet on touching them with iodide of potassium. They were proved to be corrosive sublimate. The compound was then allowed to dry, when it formed a horny translucent mass. This became readily dissolved in boiling concentrated muriatic acid, giving the usual deep purple colour formed by this acid with albumen. On diluting it with water, a precipitation of albumen took place, and the liquid gave an abundant metallic deposit on fine copper gauze. When this was dried and heated in a reduction tube, well-defined globules of mercury were obtained by sublimation. One fact appears to be obvious from this experiment, namely, that admitting the antidotal compound to consist of albumen and peroxide of mercury, it does nevertheless contain some undecomposed corrosive sublimate, not separable by mere washing with cold water, nor detectable by the addition of potash to a small quantity of it, but rendered demonstrable by long boiling in water, and subsequent filtration and evaporation. It seems to be admitted by all experimentalists, that it is destitute of any active poisonous properties, and therefore well fitted to serve as an antidote for this poison.

Gluten.—Taddei has more recently recommended *gluten* as a substance which he found to be still more effectual in decomposing the poison. Barzellotti relates the following case in proof of its efficacy. Some powdered corrosive sublimate had been accidentally mixed with calomel in an apothecary's shop, and one of the assistants, by mistake, swallowed eight grains of the poisonous mixture. He was immediately aware, from the taste, that he had swallowed the poison, and some gluten being kept ready prepared in the shop, this was immediately administered to him, mixed with water. Vomiting and purging followed, and in the vomited matters portions of a dense white substance were seen, which were supposed to be a compound of calomel and gluten. He was soon restored, although he suffered for some days from swelling of the tonsils and difficulty of swallowing. (Medicina Legale, ii. 111.) Gluten may be prepared by washing flour in a linen bag, under a current of water. Should the case be urgent, the flour may be at once exhibited in the form of paste mixed with water. Gluten may often be obtained in this way, when albumen is not at hand. These remedies cannot be expected to be always successful; the sooner they are exhibited, the greater is the hope of success. Dr. Buckler, of Baltimore, relates the case of a young man who took about fifty-five grains of corrosive sublimate in a state of perfect solution; and albumen to the amount of a quart was afterwards administered to him, but he nevertheless died on the eighth day.

Galvanic antidote.—Dr. Buckler has lately recommended the employment of a galvanic antidote,—namely, a mixture of fine gold-dust and iron-filings diffused in water, containing gum, and slightly acidulated. (Lancet, Jan. 22, 1842.) It has been found that a solution of corrosive sublimate is precipitated entirely in the form of mercury when gold-dust and iron-filings are added to it; but in practice, this mixture has entirely failed as an antidote. Orfila's experiments are decidedly adverse to its employment. (Ann. d'Hygiène, Oct. 1842.) He has found that when given in large quantity, it has not prevented animals dying within the usual time from the effects of the poison.

Hydrated protosulphuret of iron.—More recently, M. Mialhe has strongly recommended the hydrated protosulphuret of iron, which, while it is inert, produces with corrosive sublimate, protochloride of iron and bisulphuret of mercury. The protosulphuret may be made by adding hydrosulphuret of ammonia to a solution of protosulphate of iron, and washing the black precipitate without exposure to air. Orfila has lately tried some experiments with it on animals, and he has found that when exhibited speedily after the poison has been taken, it possesses even greater antidotal powers than albumen. Further observations on the human subject are, however, required, in order to confirm its efficacy as an antidote.

Protochloride of Tin.—M. Poumet has recently proposed the protochloride of tin should be used as an antidote to corrosive sublimate. The account of his experiments and results will be found in the Annales d'Hygiène, 1845, pp. 181 and 408. He considers it to be more efficacious than albumen: for, while in M. Orfila's experiments with albumen seven animals died out of twelve, only seven died out of twenty-three in employing the protochloride of tin. The conclusions at which M. Poumet has arrived are—1. That dogs are killed by doses of fifteen grains, seven grains, and even one grain and a half of corrosive sublimate, when the power of vomiting is not artificially impeded. 2. That thirty grains of the protochloride of tin, in solution, may be given to dogs without risk. They speedily recover from its effects, even when the jaws are tied in order to prevent vomiting. 3. The black precipitate (metallic mercury,) and supernatant liquid (an acid solution of perchloride of tin,) resulting from the reaction of the salt of tin on corrosive sublimate, are not poisonous. 4. A solution of two parts of protochloride injected into the stomach *immediately* after the corrosive sublimate had been swallowed, instantly and completely neutralized the deleterious properties of the mercurial salt, even when vomiting was prevented by a ligature on the jaws. 5. This successful result was obtained in two cases out of eight when the salt of tin was taken, not immediately, but a quarter of an hour after the administration of the antidote. M. Poumet therefore concludes that the protochloride is a real antidote; and he has no doubt that it would act equally well in counteracting the effects of the sulphate or nitrate of mercury.

There can be no doubt that the chemical action of the alleged antidote will render corrosive sublimate in a state of solution inert; but if the poison be in a solid form, the effect is very slow, and only partial. In the majority of M. Poumet's experiments, a solution of the poison was used. In a practical review, the results were not very favourable to the alleged antidote. The experiments which refer to the *immediate* admixture of the antidote with the poison appear to me to be quite irrelevant; and the longest period after the poison was swallowed at which the antidote was given, was a *quarter of an hour*; and even then, out of eight cases *six* proved fatal! so that it does not seem easy to refer the recovery of the other two to the mere effect of the antidote. The great want of success, even under these very favourable circumstances, would lead to but little hope of benefit when the poison had been swallowed in the solid state, or when it had been in the stomach for half an hour or an hour before the patient was seen. These very unfavourable results render it unnecessary to discuss the question, whether in the treatment of mercurial poisoning, we are justified in substituting one irritant poison for another, when, by the use of it, only one person out of four might by possibility be saved! M. Poumet states, he has found that the protochloride of tin is not poisonous; but there are a sufficient number of facts on record to show that it acts as an acid irritant poison; and he himself recommends that magnesia and milk should be given as antidotes to the antidote! Take the case of a person swallowing two drachms of corrosive sublimate in powder, or loosely diffused in a liquid. Would any medical practitioner be justified in administering half an ounce of protochloride of tin dissolved in water? It appears to me that this method of treatment would be unjustifiable. It would be as reasonable to give a large dose of sugar of lead in poisoning by an alkaline sulphuret, or of nitrate of silver in poisoning by prussic acid!

The antidotes recommended for other poisons do not require to be noticed in this place: in so far as they are likely to prove beneficial, they will be mentioned hereafter in the description of each poison.

There is, beyond question, great room for improvement in the antidotal treatment of cases of poisoning. The plan which has been hitherto adopted by experimentalists for the discovery of antidotes is most unsatisfactory. Thus, with regard to arsenic, while in ninety-nine cases out of a hundred, the medical practitioner has to treat a case in which arsenic has been taken in the state of a *solid*, the experiments in reference to antidotes have been performed on a filtered *solution*;—while, in practice, the poison has been taken in drachms and ounces, the experiments have referred to a few grains;—while, again, in practice, an hour or more often elapses before a patient can be seen by a practitioner, the experiments have been directed to the actual administration of the so-called antidote in mixture with the poison, or the substance has been given only a few minutes after the poison has been taken! Even in the introduction of the oxide of iron for the treatment of poisoning by arsenic, M. Bunsen experimented with arsenic acid, an exceedingly soluble poison, scarcely ever known to be taken by the human subject; and those who have followed him, and have experimented with arsenious acid, have obtained what they considered favourable results, under conditions scarcely ever met with in practice,—*i. e.* from operating on a few grains of the poison in a state of perfect solution. That new antidotes should be frequently recommended and old ones discarded, and that the results of treatment should be most unsatisfactory, are circumstances which cannot excite surprise, when we consider the very erroneous principles upon which experimentalists have hitherto proceeded. The greater number of persons who are engaged in practice, can only take the statement of those who have devoted their attention to researches of this kind; and they may not in all cases be competent to detect the errors into which the experimentalists, in their desire to make a useful discovery, have been unconsciously led. There is, however, this very serious result—that the preparation and employment of a substance, unless as an antidote, occasion a sacrifice of time, which may make all the difference between the preservation of life and the death of the patient. It also creates a false confidence in the mind of the practitioner, and may lead to the neglect of other appropriate means of treatment. For this reason I have devoted some space to the examination of certain substances which have recently obtained great popularity as antidotes: they have been recommended to the profession on *chemical* grounds; and it is on chemical grounds that I have examined them. It is not with antidotes as with certain medicines that we are in darkness as to the *modus operandi:* therefore, when the facts are conflicting, we are bound to consider whether erroneous conclusions may not have been drawn from the simultaneous employment of other means. To take arsenic as a type, a large number of so-called recoveries from the use of oxide of iron, have occurred under circumstances which could not possibly be referred to the chemical neutralization of the poison by combination with the oxide. I shall here quote two instances, among many, to show on what *post hoc propter hoc* reasoning such cases sometimes rest; and that in the use of antidotes, as in the use of new medicines, we must not take reported cases of recoveries, as instances of successful treatment by the alleged antidote or medicine, without fairly balancing all the circumstances under which the patient was placed. The subject of the first case, a negress, swallowed *half an ounce* of white arsenic at 3 P. M., and she was not seen until *four hours* afterwards, by Dr. Feital, when she presented, in a marked degree, the irritation, nervous and gastric symptoms, characterizing arsenical poisoning. No *vomiting* had as yet occurred, nor had any effectual treatment been adopted beyond the administration of white of eggs. Ipecacuanha was first given, in half-drachm doses, until full vomiting was produced; leeches were applied to the epigastric region, and sinapisms to the extremities, with tepid baths. When vomiting had taken place, Dr. Feital prescribed hydrated oxide of iron; but as none of this was to be had, the ordinary peroxide was substituted, of which one small spoonful was given every hour in a viscid liquid. This plan was continued for twelve hours, when the alarming symptoms had subsided, leaving the patient weak and prostrated, with great sensibility in the epigastrium. The treatment was now directed against this gastric irritation, and the patient recovered on the sixth day. From the state of the patient when first seen, there could be no doubt that some absorption of the arsenic had occurred; and the report concludes, "the operation of the iron, in relieving and subduing the dynamic symptoms, was most evident, as the result showed fully satisfactory." (Med. Times, Dec. 12, 1846, 202.)

As nothing is said to the contrary, it is to be presumed that the arsenic was here taken in the usual way, *i. e.* in coarse powder. Admitting that a portion of the poison was ejected by vomiting, still it is impossible to refer the recovery of the patient to the use of oxide of iron. One small spoonful (about one hundred grains,) given at intervals of half an hour for twelve hours, would be insufficient to neutralize or counteract chemically the effects of half an ounce of the poison, especially as the oxide of iron was in a state easily proved by experiment to be decidedly unfavourable to its action. Dr. Maclagan's researches have shown that it requires twelve parts of moist oxide of iron, and *sixty parts* if it be previously dried (as it was in this case,) to remove one part of arsenic from a state of solution. (Christison on Poisons, 364.) From my own experiments, I have not the least doubt of the general accuracy of his results. The small quantity of dried oxide of iron (two and a half ounces) here given, in divided doses, in twelve hours, could then have had no perceptible effect; and the recovery of the patient, after having taken such a formidable dose of arsenic, can only be ascribed to active vomiting and great strength of constitution. The second case is perhaps even more remarkable; for it is alleged that the carbonate of iron, introduced into the stomach, chemically counteracted the poison as it existed in the blood! A man, wishing to cure himself of the itch, rubbed his skin with a solution of arsenious acid and sulphate of copper. On the following day there was thirst, with dryness in the throat, and other symptoms of arsenical poisoning. In about twenty hours he took, at intervals of half an hour, about *one drachm* of carbonate of iron, suspended in thick gruel. In two hours after he had taken the carbonate, the mouth and fauces became moist; the pain ceased, and the patient slept through the night. The next day his symptoms had become relieved. (Galtier, Toxicologie, i. 422.) If the carbonate of iron had any effect in this case, it could not have been by entering into combination with and rendering the poison insoluble in the blood. The case, like many others, proves too much, and shows what caution ought to be used in drawing inferences from the results of particular modes of treatment.

General Treatment.—When the poison is no longer in the alimentary canal, the practitioner will have to combat the physical changes produced by it, and the alarming symptoms which result from its absorption. Antidotal treatment is here of no avail: the poison is beyond the reach of substances of this kind. Our object must be to palliate symptoms by antiphlogistic measures; for recovery from the first effects produced by the irritants, is generally followed by inflammation of the alimentary canal. No general rules can be laid down, as the treatment must vary with the nature of the poison. There are no means of counteracting the effect of the poison when absorbed. In respect to arsenic, Orfila has recommended the use of diuretics, because, according to his researches, this poison is largely carried off by the renal secretions. Independently of the fact that, in arsenical poisoning, there is sometimes a complete suppression of urine, and that in other cases there is an entire want of proof that arsenic escapes more through this than through other excretions, this plan of treatment has been tried and utterly failed. (Traité des Poisons, Flandin, vol. i. 331.) The treatment of cases of poisoning in which the poison has become already absorbed and carried into the circulation, must be regarded as beyond the powers of art: recovery in these instances appears to depend rather upon strength of constitution, and the small amount of local injury produced by the substance, than upon medical treatment.

In making these remarks, it may appear that I am undervaluing the application of therapeutical agents to the expulsion of the poison by elimination. Primâ facie, a system of treatment which implies that all the poison in the body should be made to traverse the circulation with as much rapidity as possible, is unadvisable, even if experimentalists were agreed upon the channel through which the noxious substance is eliminated. It is the entrance of the poison into the blood which places life in danger, and our efforts should be directed rather to prevent than to promote its absorption. While M. Orfila contends that arsenic passes off by the kidneys, and advises diuretics, M. Flandin insists, from the results of his experiments, that it passes off by the biliary secretion from the liver, and by the cutaneous exhalation from the skin. He therefore advises the free use of purgatives, and the exhibition of magnesia and sulphuretted waters, to neutralize and render unsusceptible of re-absorption, that portion of arsenic which may be poured out upon the mucous surface of the intestines. (Des Poisons, i. 586.) When all the poison has been expelled from the alimentary canal, there can be no injury in giving diuretics or purgatives; but it is questionable whether any good would in any case result from their administration.

CHAPTER VIII.

RULES TO BE OBSERVED IN INVESTIGATING A CASE OF POISONING—WITH RESPECT TO THE PATIENT WHILE LIVING—THE INSPECTION OF THE BODY—THE EXUMATION OF BODIES—DISPOSAL OF THE VISCERA. IDENTITY OF SUBSTANCES. PRESERVATION OF ARTICLES FOR ANALYSIS. ON THE USE OF NOTES—WHEN ALLOWED TO BE USED IN EVIDENCE—WHEN INADMISSIBLE.

WHEN a practitioner is called to a case of poisoning, it is above all things necessary that he should know to what points he ought to give his attention. It is very proper that every effort should be made by him to save life when the individual is still living: but while engaged in one duty, it is also in his power to perform another, supposing the case to be one of suspected criminal poisoning,—namely, to note down many circumstances which may tend to detect the perpetrator of the crime. There is no person so well fitted to observe these points as a medical man; but it unfortunately happens, that many facts important as evidence, are often overlooked. The necessity for observing and recording them, is not perhaps generally known. A medical man need not make himself officious on such occasions, but he would be sadly unmindful of his duty as a member of society, if he did not aid the course of justice by extending his scientific knowledge to the detection of crime. It is much to the credit of the medical profession, that the crime of murder by poisoning—a form of death from which no caution or foresight can protect an individual, is so frequently brought to light, by the announcement of suspicious facts of a medical nature to magistrates and coroners; and on several occasions the highest compliments have been passed by judges, on medical practitioners who have been thus indirectly the means of bringing an atrocious criminal to the bar of justice.

The following appear to me to 'be the principal points which demand the attention of a medical jurist in all cases of suspected poisoning:—1. With respect to

SYMPTOMS.—1. The time of their occurrence,—their nature. 2. The exact period at which they were observed to take place after a meal, or after food or medicine had been taken. 3. The order of their occurrence. 4. Whether there was any remission or intermission in their progress, or whether they continued to become more and more aggravated until death. 5. Whether the patient had laboured under any previous illness. 6. Whether the symptoms were observed to recur more violently after a particular meal, or after any particular kind of food or medicine. 7. Whether the patient has vomited:—the vomited matters, if any (especially those *first* ejected,) to be procured:—their odour, colour, and acid or alkaline reaction noted,—as well as their quantity. 8. If none be procurable, and the vomiting have taken place on the dress, furniture, or floor of the room,—then a portion of the clothing, sheet, or carpet, may be cut out and reserved for analysis:—if the vomiting have occurred on a deal floor, a portion of the wood may be scraped or cut out:—or if on a stone pavement, then a clean piece of rag or sponge soaked in distilled water, may be used to remove any traces of the substance. [Some years since, an animal was poisoned by arsenic. None of the poison could be detected in the stomach, but it was easily found in a portion of deal floor, rendered humid by the liquid matters which the animal had vomited during the night.] The vessel in which vomited matters have been contained will often furnish valuable evidence, since heavy mineral poisons fall to the bottom, or adhere to the sides of the vessel. 9. Endeavour to ascertain the probable nature of the food or medicine last taken, and the exact time at which it was taken. 10. Ascertain the nature of *all* the different articles of food used at a meal. 11. Any suspected articles of food, as well as the vomited matters, to be as soon as possible sealed up in a clean glass vessel, labelled, and reserved for analysis. 12. Note down in their own words, all explanations voluntarily made by parties present, or who are supposed to be concerned in the suspected poisoning. 13. Whether more than one person partook of the food or medicine:—if so, whether all these persons were affected, and how. 14. Whether the same kind of food or medicine had been taken before by the patient or other persons without ill effects following. In the event of the *death* of the patient, it will be necessary for a practitioner to note down—15. The *exact time* of death; and thus determine how long a period the person has survived after having been first attacked with the symptoms. 16. Observe the attitude and position of the body. 17. Observe the state of the dress. 18. Observe all surrounding objects. Any bottles, paper packets, weapons, or spilled liquids lying about, should be collected and preserved. 19. Collect any vomited matters near the deceased. Observe whether vomiting has taken place in the recumbent position or not. If the person have vomited in the erect or sitting posture, the front of the dress will commonly be found covered with the vomited matters.

Inspection of the Body.—20. Note the external appearances of the body, whether the surface be livid or pallid. 21. Note the state of the countenance. 22. Note all marks of violence on the person, or discomposure of the dress,—marks of blood, &c. 23. Observe the presence or absence of warmth or coldness in the legs, arms, abdomen, mouth, or axillæ. 24. The presence of rigidity or cadaverous spasm in the body. To give any value to the two last-mentioned characters, it is necessary for the practitioner to observe the nature of the floor on which the body is lying, whether this be clothed or naked, young or old, fat or emaciated. All these conditions create a difference, in respect to the cooling of the body and the access of rigidity. 25. If found dead—When was the deceased last seen living, or known to have been alive? 26. Note all circumstances leading to a suspicion of suicide or murder. 27. The time after death at which the inspection is made. 28. Observe the state of the abdominal viscera. If the stomach and intestines be found inflamed, the seat of inflammation should be exactly specified; also all marks of softening, ulceration, effusion of blood, corrosion, or perforation. The stomach should be removed and placed in a separate vessel, a ligature being applied at the cardiac and pyloric ends. 29. The contents of the stomach should be collected in a clean *graduated* vessel:—notice *a* the quantity, *b* the odour tried by several persons, *c* the colour, *d* acid or alkaline reaction, *e* presence of blood, mucus, or bile, *f* presence of undigested food; and here it may be as well to observe, that the presence of farinaceous matters (bread) would be indicated by the addition of iodine water, if the contents were not alkaline—of fat, by heat; *g* other special characters. 30. The contents of the duodenum should be separately collected, ligatures being applied to it. 31. Observe the state of the large intestines, especially the rectum, and note the condition of their contents. The discovery of hardened fæces in the rectum would prove that diarrhœa had not existed recently before death. In one case which I had lately to examine, this became a question of considerable importance. 32. The state of the larynx, fauces, and œsophagus,—whether there be in these parts any foreign substances, or marks of inflammation or corrosion. This is of essential importance, as it throws light upon the question, whether the poison swallowed was irritant or corrosive, and whether it had or had not a local chemical action. 33. The state of the thoracic viscera:—all morbid changes noted. 34. The state of the brain. 35. The condition of the genital organs should be examined, as in the female, poison has been sometimes introduced into the system by the vagina.

Such are the points to which, in the greater number of cases of suspected poisoning, a medical jurist should attend. By means of these data, noted according to the particular case to which they are adapted, he will in general be enabled, without difficulty, to determine the probable time of death, the probable cause of death, and the actual means by which death was brought about. He may thereby have it in his power also to point out the dish which had contained the poison, if the case be one of poisoning; and to throw light upon any disputed question of suicide or murder in relation to the deceased. Many cases of poisoning are obscure, owing to these points not having been attended to in the first instance.

I have not considered it necessary to enter into any details respecting the mode of performing an inspection. This the practitioner will have acquired during his study of anatomy; and the only essential points in addition to those mentioned, are—1. To examine all the important organs for marks of natural disease; and 2. To note down any unusual pathological appearances, or abnormal deviations; although they may at the time appear to have no bearing on the question of poisoning. It is useful to bear in mind on these occasions, that the body is inspected, not merely to show that the individual has died from poison, but to prove that he has not died from any natural cause of disease. Medical practitioners commonly direct their attention exclusively to the first point; while lawyers, who defend accused parties, very properly direct a most searching examination to the last mentioned point, *i. e.* the healthy or unhealthy state of those organs which are essential to life, and with which the poison has not probably come in contact. The most usual causes of sudden death commonly have their seat in the brain, the heart and its great vessels, and in the lungs. Marks of effusion of blood, congestion, inflammation, suppuration, or a diseased condition of the valves of the heart, should be sought for and accurately noted, whatever may be the condition of the abdominal viscera. It has also been recommended that an examination of the spinal marrow should be made. If the cause of death be very obscure after the general examination of the body, there may be some reason for inspecting the condition of this organ; but except in the case of *Tawell*, I have never known the omission to examine the spinal marrow, made a serious part of the defence.

Exhumation of Bodies.—Sometimes the inspection of a body is required to be made long after interment. So long as the coffin remains entire, there may be the expectation of discovering certain kinds of mineral poison in the organs; but decomposition may have advanced so far as to destroy all pathological evidence. The inspection is in such cases commonly confined to the abdominal viscera. The stomach is often found so thinned and collapsed, that the anterior and posterior walls appear to form only one coat. This organ should be removed, with the duodenum, and ligatures applied to each. The liver and the spleen should also be removed, in order that they may, if necessary, be separately analysed. If poison be not found in these viscera, it is not likely that it will be discovered in the body. It has been recommended that a portion of earth immediately above and below the coffin should be removed for analysis, as it may contain arsenic; but this appears to me to be an unnecessary piece of refinement, in those cases where the coffin is entire, or where the abdominal parietes still cover the viscera. When decomposition is so far advanced as to have led to a mixture of the earth with the viscera, and the poison is found in minute quantity in the tissues only, the case should be regarded as beyond the reach of analysis. Upon such hyperchemical views it might be fairly objected, that arsenic always exists in the iron and brass nails and ornaments which are used in a coffin; and this arsenic is just as likely to furnish a valid objection to medico-legal researches as that which is said to be a constituent of all soils in which oxide of iron abounds!

It is important that the viscera taken from a body which has been long in the grave should be sealed up immediately. They should not be allowed to come in contact with any metal, nor with any surface except that of clean glass, porcelain or wood. It has been recommended that they should be washed with chloride of lime, or placed in alcohol; but this is decidedly improper: the use of any preservative chemical liquid would not only embarrass the future analysis, but would render a special examination of an unused portion of the liquid necessary—the identity of which would have to be unequivocally established. Preservation from air in clean glass vessels, with well-fitted corks, covered with skin, or what is still better, sheet-caoutchouc, is all that is required in practice.

Identity of substances.—It is necessary to observe, that all legal authorities rigorously insist upon proof being adduced of the *identity* of the vomited matters or other liquids taken from the body of a deceased person, when poisoning is suspected. Supposing that during the post-mortem examination, the stomach and viscera are removed from the body, they should never be placed on any surface, or in any vessel, until we have first ascertained that the surface or vessel is perfectly *clean*. If this point be not attended to, it will be in the power of counsel to raise a doubt in the minds of the jury, as to whether the poisonous substance might not have been accidentally present in the vessel used. This may be regarded as a very remote presumption; but nevertheless, it is upon technical objections of this kind, that acquittals follow, in spite of the strongest presumptions of guilt. This is a question for which every medical witness should be prepared, whether he be giving his evidence at a coroner's inquest, or in a court of law. Many might feel disposed to regard matters of this kind as involving unnecessary nicety and care; but if they be neglected, it is possible that a case may be at once stopped; so that any care bestowed upon the chemical analysis by the practitioner, will thus have been thrown away. Evidence of the presence of the poison in the contents of a stomach was once rejected in a court of law, because they had been hastily thrown into a jar borrowed from a neighbouring grocer's shop; and it could not be satisfactorily proved that the jar was clean and entirely free from traces of poison (in which the grocer dealt) when used for this purpose. When the life of a human being is at stake, as in a charge of murder by poisoning, the slightest doubt is always very properly interpreted in favour of a prisoner.

Not only must clean vessels be used for receiving any liquid destined for subsequent chemical analysis; but care must be taken by the practitioner that the identity of a substance is preserved, or the most correct analysis afterwards made, will be inadmissible as evidence. The suspected substance, when once placed in his hands, should never be let out of his sight or custody. It should be kept sealed under his private seal, and locked up while in his possession, in a closet to which no other person has a key. If he has once let it out of his hands, and allowed it to pass through the hands of several other persons, then he complicates the evidence for the prosecution, by rendering it indispensible for these parties to state under what circumstances it was placed, while in their possession. The exposure of a suspected substance on a table, or in a closet or room, where many have access, may be fatal to its identity; for the chemical evidence, so important in a criminal investigation, will probably be altogether rejected by the court. A few years ago, a case was tried on the Norfolk circuit, in which the analysis of the matters vomited by a person poisoned by arsenic, was not admitted as evidence against the prisoner, because the practitioner had left them in the keeping of two ignorant women; and these women had allowed the vessel containing the suspected liquid (which was proved to contain arsenic) to be exposed in a room open to the access of many persons. In another case, tried at the Old Bailey sessions in 1835, the analysis of some suspected liquids was not allowed in evidence, because the practitioner, who lived in the country, and was unwilling to take upon himself the responsibility of analysing them, had sent them up to town by a carrier, to be examined by a London chemist. If closely sealed by a private seal, and this be observed by the receiver to be unbroken, before he proceeds to the analysis—this mode of transmission will not probably be objected to. When any article (*e. g.* a stomach or other viscus) is reserved for analysis, care should be taken to attach immediately to it, or the vessel containing it, a label upon which is plainly written in ink, the name of the deceased and the date of removal, including the day of the week and month. This is especially necessary when there are two or more articles for analysis. I have known the greatest inconvenience result from the neglect of this simple precaution.

Preserving articles for analysis.—In removing viscera or liquids from the body, and reserving them for analysis, it is necessary to observe certain precautions. A clean vessel with a wide mouth should be selected: it should be only sufficiently large to hold the viscus or liquid (the less air remaining in it the better;) it should be secured by a closely fitting cork, covered with fine skin or bladder. Another piece of skin should then be tied over the mouth, or for this sheet-caoutchouc may be substituted with advantage. This should be covered with tin foil, and lastly with white leather. In this way any loss by evaporation or decomposition is prevented, and the viscera may be preserved (in a cool place) for some time. If the mouth of the vessel be too wide for a cork, the other articles cannot be dispensed with. Paper only should not be used; I have known the post-mortem appearances of the viscera of an infant, suspected to have died from poison, entirely destroyed by drying, from the evaporation which took place through the layers of paper with which the vessel in which they were contained, was covered. The practitioner should bear in mind that all these matters are likely to come out in evidence; and whatever is worth doing at all, is worth doing well. No antiseptic should be used. The addition of alum, alcohol, or chloride of lime to the viscera, may seriously embarrass the analysis. (See post, CHEMICAL ANALYSIS.)

ON THE USE OF NOTES.—It has already been recommended as a rule in these criminal investigations, that a practitioner should make notes of what he observes in regard to symptoms, post-mortem appearances, and the results of a chemical analysis. From the common forms of law in this country, an individual charged with the crime of poisoning may remain imprisoned, if at a distance from the metropolis, for some months before he is brought to trial. It is obvious, however clear the circumstances may at the time appear to the practitioner, that it will require more than ordinary powers of memory to retain for so long a period, a distinct recollection of all the facts of the case. If he be unprovided with notes, and his memory be defective, then the case will turn in favour of the prisoner, for he will be the party to benefit by the neglect of the witness. In adopting the plan here recommended, such a result may be easily prevented. It may be remarked, that the law relative to the admissibility of notes or memoranda in evidence is very strict, and is rigorously insisted on by the judges. In order to render such notes or memoranda admissible, it is indispensably necessary that they should be taken on the spot at the time the observations are made, or as soon afterwards, as practicable. It is not necessary to their admissibility as evidence, that the observations should be written down by the practitioner himself, provided they are made under his immediate inspection at the time, or at his suggestion, and are soon afterwards looked over and corrected by him. Thus, whenever, at a trial, a medical witness produces notes for reference during his examination, the question is invariably put to him as to when the notes were made. Their admissibility depends upon his answer. Many examples might here be cited of the rejection of notes, made by medical witnesses, owing to a non-observance of these points. On the trial of *Sir A. Gordon Kinloch* at Edinburgh, for the murder of his brother, the surgeon was about to give his evidence respecting the wound of which the deceased had died, from notes made some time after the event, when he was stopped by the judge, who explained to him the law on the subject. The reason why the law so rigorously excludes the admission of memoranda in evidence, made at a distance of time, appears to be this: it prevents the possibility of all fraud or collusion on the part of the witnesses, either to favour or injure the prisoner; for a connected story might, it is presumed, by such means, be so made up at a distance of time, as to defy the ingenuity of counsel on either side to make out the deceit. The notes used by a witness should be original, and not a *copy* of notes made by another. A copy of notes, except under very peculiar circumstances, is not admissible as evidence.

There is another rule of law with respect to the use of notes or memoranda in evidence, which is not perhaps so generally known to medical practitioners; but it is essential that it should be observed. The notes may have been fairly made on the spot in the manner required by law; but when a witness is about to refer to them, he will probably be asked whether he is using them for the purpose of refreshing his memory, or whether he is about to speak only from what is written on the paper, without having any precise recollection on the subject. If he is referring to them for some fact which he has altogether forgotten, then the notes are pro tanto inadmissible as evidence; for it has been held by our judges, that notes can only be used in evidence for the purpose of refreshing the memory on a fact *indistinctly remembered:* they are not permitted to be used for the purpose of reviving impressions entirely forgotten. The most eminent legal writers lay it down, that if there be any single point in the notes, which the witness does not recollect, except that he finds it there written, such point is not evidence. Notes are only allowed to assist recollection, not to convey information.

On a late trial for poisoning, the medical witness, after having detailed the action of some tests, which he had employed in the detection of the poison, referred to his notes before giving the results of other experiments. Upon being asked when the notes were made, he answered satisfactorily by stating, that they were taken at the time of the observations. The counsel then asked the witness, whether he used the notes to refresh his memory, or whether he had forgotten the subject, and was about to speak only from what was written on the paper. The witness said, that his memory was bad; that some time had elapsed, and he had entirely forgotten the results of these experiments. It was then objected, that the results could not be given in evidence, since the witness could only speak to the facts from the memorandum which he held in his hand. The objection was admitted by the judge, and the evidence from the analysis was rejected. The reason for a rigorous adherence to this rule of law is not very apparent. In applying it to medical evidence, it must often operate in such a way, as to give an undue advantage to a criminal. Many witnesses who are summoned to give evidence in courts of law, are not much accustomed to the analysis of poisons; and probably may never have performed the usual experiments, until that particular case occurred to draw their attention to the subject. It is well known to practised analysts, that numerous tests are required in order to show what a suspected substance is, as well as what it is not;—that the action of these tests is sometimes attended with results not easily remembered; and it can be no imputation upon those who are less practised in toxicology, that they should not be able to retain for many months together, the whole of the results of such experiments. We might suppose that the making of a record at the time, would be sufficient to render the evidence admissible; nor does it appear how the adoption of such a rule could ever injure a really innocent person. From the rejection of notes on this ground, in the case above quoted, it would seem that good medical evidence, to be available in a court of law, must in many cases depend upon the possession of an excellent memory by the witness. But many men, equally well informed, and fitted to act as witnesses, are not equally endowed with this faculty. If justice cannot be safely administered without enforcing such a rule, then it follows, that only those persons who have powerful memories should be selected to act as witnesses. But on the other hand, with an excellent memory, there may not be that mental capacity which renders a man well fitted for observing those medical facts from which good evidence must always be drawn. These remarks on the taking of notes, and the method of using them in a Court of law, have here been made in reference especially to cases of criminal poisoning; since they are, perhaps, more frequently required in these, than in other medico-legal cases. It will be understood, however, that they apply with equal force to every medico-legal investigation in which the practitioner may be concerned.

CHAPTER IX.

ON THE EVIDENCE OF POISONING IN THE DEAD BODY—PERIOD AT WHICH POISONS PROVE FATAL—CHRONIC POISONING—ACCUMULATIVE POISONS—POST-MORTEM APPEARANCES PRODUCED BY THE DIFFERENT CLASSES OF POISONS—REDNESS OF THE MUCOUS MEMBRANE—MISTAKEN FOR INFLAMMATION—ULCERATION AND CORROSION—SOFTENING—PERFORATIONS OF THE STOMACH—OF THE INTESTINES FROM DISEASE—BY WORMS—SPECIES OF WORMS FOUND IN THE BODY.

SUPPOSING that the person is dead, and we are required to determine whether the case be one of poisoning or not, we must, in the first instance, endeavour to ascertain all the particulars which have been discussed in the last chapter, as indicative of poisoning in the living subject. Should the deceased have died from poison, the circumstances of the attack, and the symptoms preceding death, ought to correspond with the characters already described; and in these investigations it is well to bear in mind the following rule:—There is no one symptom or pathological condition which is peculiar to poisoning; but at the same time there is no disease which presents *all* those characters met with in a special case of poisoning. The additional evidence to be derived from the *death* of the person may be considered under the following heads:—

1. THE TIME AT WHICH DEATH TAKES PLACE AFTER THE FIRST OCCURRENCE OF SYMPTOMS.—This question it is necessary to examine, because the more common poisons, when taken in fatal doses, generally produce their fatal effects within certain periods of time. By an attention to this point, we may, in some instances, be enabled to negative a charge of poisoning, and in others to form an opinion of the kind of poison which has been taken. In a Court of law a medical practitioner is often required to state the usual *period of time* within which poisons prove fatal. It is to be observed, that not only do poisons differ from each other in this respect, but the same substance, according to the form or quantity in which it has been taken, will differ in the rapidity of its action. A large dose of strong prussic acid, i. e. from half an ounce to an ounce, may

destroy life in less than two minutes. In ordinary cases of poisoning by this substance, a person dies, *i. e.* all signs of life have commonly ceased, in from ten to twenty minutes:—if he survive half an hour, there is some hope of recovery. In the cases of the seven Parisian epileptics, accidentally poisoned by this acid, the first died in about twenty minutes, the seventh survived three quarters of an hour.—(See post, Prussic Acid.)—Oxalic acid, one of the most energetic of the common poisons, when taken in a dose of from half an ounce to an ounce, may destroy life in from ten minutes to an hour: if the poison be not perfectly dissolved when swallowed, it is a longer time in proving fatal. The strong mineral acids, in poisonous doses, destroy life in about eighteen or twenty-four hours. Arsenic, under the form of arsenious acid (white arsenic,) operates fatally in from eighten hours to three or four days. It has, however, in more than one instance, killed a person in two hours; although this is by no means common. Opium, either as a solid or under the form of laudanum, commonly proves fatal in from six to twelve hours; but it has been known, in several instances, to destroy life in less than three hours: those who survive the effects of this poison for twelve hours, are considered to have a fair chance of recovery. This must be understood to be merely a statement of the average results, as nearly, perhaps, as we are warranted in giving an opinion: but the medical jurist will of course be aware, that the fatal period may be protracted or shortened, according to all those circumstances which have been elsewhere stated to affect the action of poisons.

There are various forms which this question may assume in a Court of law:—the death of a party, alleged to have taken poison, may have occurred too rapidly or too slowly to justify a suspicion of poisoning. The following case may serve as an illustration:—A woman of the name of *Russell* was tried and convicted at the Lewes Summer Assizes, in 1826, for the murder of her husband, by poisoning him with arsenic. The poison was detected in the stomach; but the fact of poisoning was disputed by some medical witnesses, for this among other reasons—that the deceased had died *three* hours after the only meal at which the poison could have been administered to him. The authority of Sir A. Cooper and others, was cited to show, that according to their experience, they had never known a case to prove fatal in less than seven hours. This may well have been; but, at the same time there was sufficient authority on the other side, to establish that some cases of arsenical poisoning had actually proved fatal in three or four hours. So far as this objection was concerned, the prisoner was very properly convicted.

On the medical question raised at this trial, I may observe, that within the last few years two distinct cases have occurred where the individual died certainly within two hours after taking this poison; and several instances have been reported since the trial, in which death took place in from three to four hours after the administration of arsenic. It seems extraordinary in the present day, that any attempt should have been made by a professional man to negative a charge of criminal poisoning upon so weak a ground as this; but we must remember, that this opinion was expressed twenty years ago, when the subject of toxicology was but little understood. It is quite obvious, that there is nothing, so far as we know, to prevent arsenic from destroying life in an hour. A case will be hereafter related, in which death took place, most probably from arsenic, in half an hour. These matters can only be settled by a careful observation of numerous cases, and not by any à priori reasoning or reference to personal experience.

In all instances of sudden death, there is generally a strong tendency on the part of the vulgar to suspect poisoning. They never can be brought to consider, that persons may die a natural death suddenly, as well as slowly; or, as we shall presently see, that death may really take place slowly, as in cases of disease, and yet be due to poison. This prejudice continually gives rise to the most unjust suspicions of poisoning: a case illustrating this has already been given (ante, p. 60.) One of the means recommended for distinguishing narcotic poisoning from apoplexy or disease of the heart, is the difference in the rapidity with which death takes place. Thus, apoplexy or disease of the heart may prove fatal either instantly or within an hour. The only common poison likely to operate with such fatal rapidity is prussic acid. But when this is the cause of death, some traces of poison will be found at hand, except in a case of murder (*Reg.* v. *Tawell*, Bucks Lent. Ass. 1845.) Poisoning by opium is commonly protracted for five or six hours. This poison has never been known to destroy life instantaneously, or in a few minutes. I here exclude strychnia, as well as morphia and its salts; for these are poisons out of common reach. Thus, then, it may happen, that death will occur with such rapidity, as to render it impossible to attribute it to narcotic poison under the circumstances.

The following case, reported by Anglada is of some interest:—A lady in perfect health, while supping with her husband and family, complained, after having taken two or three mouthfuls,—of severe pain in the region of the heart. She fell back in her chair and died instantly. The parties not having lived on the best of terms, the husband was openly accused of having been accessary to the poisoning of his wife,—a circumstance which was rendered still more probable in the opinion of his neighbours, by the fact that his wife had lately made a holographic will in his favour. One of his servants, with whom he was said to live in adultery, was arrested, and a paper containing a white powder was found in her possession. The husband endeavoured to compromise the affair by offering to give up the will. Here, then, were strong moral presumptions of death from poisoning. Three surgeons (experts!) were appointed to examine the body. They opened the abdomen, and observing some green spots in the stomach, (produced, as it afterwards appeared, by imbibition from the gall-bladder,) pronounced an opinion that the organ was in a gangrenous state from the effects of some corrosive poison. Some doubt arising on the correctness of this view, four other surgeons were directed to re-examine the body. They found that the stomach had not even been opened,—and that its mucous membrane, as well as that of the intestines, was perfectly healthy! It contained a small quantity of undigested food, which was free from any trace of poison. The deceased had died from natural causes. The white powder found in the possession of the servant was nothing more than white sugar. Had the usual effects of poisons been attended to by the parties who were first called to give evidence in this case, it is obvious that no charge of poisoning could have been made with any shadow of probability. The deceased died suddenly:—there is no common poison which acts so rapidly except strong prussic acid in a very large dose. It was very unlikely that this should have been administered in solid food. Besides, there was no vomiting before death; hence it followed, that if poison were the cause, the whole of it must have been found in the viscera; but none was discovered.

The great utility of the coroner's inquest in England, when the cause of death is properly investigated, is seen in its removing such suspicions.

Sometimes the only medical evidence on which we may have to speak to the fact of poisoning, will be the *duration* of the case. There is, then, of course, great difficulty in forming an opinion; but we may generally be able to say whether the time which the party survived after his first illness, was or was not compatible with any known form of poisoning; and also in some instances to determine the probable nature of the poison, if any were really taken by the deceased. The following case is in some respects interesting: it occurred a few years since in the metropolis, to a gentleman who was then one of my pupils, and consulted me respecting it:—A woman, by occupation a laundress, was found lying dead on the floor of her kitchen. The deceased was about forty years of age, of spare make, and of temperate habits. When first seen, she was lying on the stone floor in a curved position, on her right side, her right cheek being in a plate which contained four ounces of light brown liquid, mixed with mucus. There was no doubt that this had issued from her mouth by vomiting. The countenance did not express anxiety or pain, but the lips were somewhat drawn in. There was no suffusion about the eyes. The surface was slightly livid. She was but lightly clothed. Some bread and meat were found at a distance; and on a shelf, in a distant part of the kitchen, some bottles of oil, which had evidently not been touched. No bottle containing poison, nor any weapon, was found about the place. There were no marks of violence on the person. The woman had been a little indisposed for about a fortnight previously, and had taken medicine; she, however, was well enough to work. She was last seen alive about six o'clock in the afternoon, when she appeared in her usual health, and was heard moving about in her room at half-past nine the same evening:—she was found dead twelve hours afterwards, *i. e.* at half-past nine the following morning. As the body was quite cold, and the members perfectly rigid when first seen, it is reasonable to presume that the deceased had been dead at least ten hours—the weather not being very cold at the time. Hence it is probable that she died about half-past eleven o'clock at night, *i. e.* about *two hours* after she had been heard moving in her room, and about five or six hours after she was last seen in her usual health and spirits. From these facts, it was considered very unlikely that she should have died from poison. The only poisons which could be suspected, to prove so rapidly fatal, were prussic acid, opium, or oxalic acid. Had prussic acid been the cause, it would have been easily discovered by the odour, as well as by some of the poison being found near. Had opium or oxalic acid been the cause of death, a portion of either of these bodies would have been found in the liquid vomited in the plate, but this contained no trace of any kind of poison. The deceased could not have died very suddenly, since she had had time to take a plate from the sideboard and lie down with it. Had she fallen with the plate in her hand, it must have been broken on the stone floor. Besides, as there were no marks of vomiting on the front of her dress or elsewhere in the room, it is pretty certain that she must have vomited while in the recumbent position. At the inquest, no post-mortem examination was required by the coroner or jury; and therefore it is difficult to say what was really the cause of death. The gentleman who conducted the case, very properly inferred that the deceased had not died from poison.

Chronic poisoning.—When a poison destroys life rapidly, it is called a case of *acute* poisoning, to distinguish it from the *chronic* form, *i. e.* where death takes place slowly. Chronic poisoning is not a subject which often requires medico-legal investigation. Most poisons are capable, when their effects are not rapidly manifested, either from the smallness of the dose or from timely treatment, of slowly undermining the powers of life, and killing the patient by producing emaciation and exhaustion. This is sometimes observed in the action of arsenic and corrosive sublimate, but it has been remarked also in cases of poisoning by the mineral acids and caustic alkalies. Death is here an indirect consequence:—stricture of the œsophagus is induced, or the lining membrane of the stomach is destroyed and the process of digestion impaired, —a condition which leads to emaciation and death. The time at which these indirect effects will prove fatal, is of course liable to vary. A person has been known to die from a stricture of the œsophagus brought on by sulphuric acid, *eleven months* after the poison was swallowed; and there is no reason to doubt that instances may occur of a still more protracted nature. In these cases of *chronic poisoning*, there is considerable difficulty in assigning death exclusively to the original action of the poison, since the habits of life of the party,—a tendency to disease, and other circumstances, may have concurred to accelerate or produce a fatal result. To connect a stricture of the œsophagus with the act of poisoning by a mineral acid, it is necessary to show that there was no tendency to this disease before the acid was administered:—that the symptoms appeared soon after the first effects of the poison went off:—that these symptoms continued to become aggravated until the time of death; and that there was no other cause to which death could with any probability be referred. These remarks apply equally to the indirectly fatal effects of any poison,—such, for instance, as the salivation occasionally induced by corrosive sublimate, when the acute symptoms of poisoning by that substance, have passed away. It has been stated, that chronic poisoning is not a subject commonly requiring a criminal investigation. Two cases have, however, come before our tribunals, in which the facts connected with this form of poisoning, were of some importance. I allude to those of *Miss Blandy*, tried at Oxford, in 1752, for the murder of her father by arsenic; and of a woman named *Butterfield*, tried at Croydon, in 1775, for the murder of a Mr. Scawen, by administering corrosive sublimate. In most cases, murderers destroy life by administering poison in very large doses; but in these instances, small doses were given at intervals, —a fact which led to great medical doubt of the real cause of the symptoms before death. It is, however, very rare to hear of this form of poisoning.

It has been already remarked, that some poisons have what is called an *accumulative* property, *i. e.* they may be administered for some time in small doses without producing any marked effects; but they will, perhaps, after a certain period, suddenly and unexpectedly give rise to violent symptoms, affecting the life of a person. This peculiar mode of action has been witnessed more in medical practice than in cases of attempts to poison; hence it is not a subject of much importance to a medical jurist. Foxglove (digitalis) is said to possess this property; and it has been remarked that, on more than one occasion, persons to whom this medicine has been repeatedly administered in small doses have suddenly died, probably from the accumulative properties of the poison. The same effect has been noticed in the case of other poisons.

2. EVIDENCE FROM POST-MORTEM APPEARANCES.—One of the chief means of determining whether a person has died from poison, is an examination of the body after death. In relation to *external* appearances, there are none indicative of poisoning upon which we can safely rely. It was formerly supposed, that the bodies of persons who were poisoned, putrefied more readily than those of others who had died from natural disease; and evidence for or against poisoning, was at one time drawn from the external appearance of the body. This is now known to be an error; the bodies of persons poisoned are not more rapidly decomposed, cæteris paribus, than those of others who have died a sudden and violent death from any other cause whatever.

Irritant poisons act chiefly upon the stomach and intestines, which they irritate, inflame, and corrode. We may likewise meet with all the consequences of inflammation, such as ulceration, perforation and gangrene. Sometimes the coats of the viscera are thickened, at other times thinned and softened by the action of an irritant.

Narcotic poisons do not commonly leave any well-marked post-mortem appearances. The stomach and intestines present no unnatural changes. There is greater or less fulness of the cerebral vessels; but even this is often so slight as to escape notice, unless attention be particularly directed to the brain. Extravasation of blood is rarely found.

The *Narcotico-irritants* affect either the brain or the alimentary canal, and commonly both, according to their peculiar mode of action.

It is important to bear in mind, that both irritants and narcotics may destroy life without leaving any appreciable changes in the body. To such cases as these, the remarks about to be made do not apply. The proof of poisoning must, then, be derived entirely from other sources. Any evidence derivable from the appearances in the body of a person poisoned, will be imperfect unless we are able to distinguish them from those analogous changes, often met with as the results of ordinary disease. These are confined to the mucous membrane of the stomach and bowels. They are redness, ulceration, softening, and perforation. All of these conditions may depend upon disease, as well as upon the action of irritant poisons.

REDNESS.—It is a main character of the irritants to produce redness of the mucous membrane of the stomach and small intestines. This redness, when first seen, is usually of a deep crimson colour, becoming brighter by exposure to air. It is sometimes diffused over the whole mucous membrane:—at other times it is seen in patches over the surface of the stomach. It is sometimes met with at the smaller, but more commonly at the larger extremity of the organ; and then, again, we occasionally find that the rugæ or prominences only of the mucous membrane present this red or inflamed appearance.

Redness of the mucous membrane may, however, be due to gastritis or gastro-enteritis; and in order to assign the true cause, it will be necessary to have an account of the symptoms preceding death, or some proof of the existence of irritant poison in the contents of the stomach or the tissues of the body. In this respect the following case (*Reg.* v. *Hunter*, Liverpool Spring Assizes, 1843,) is of some interest. A woman was charged with having poisoned her husband by arsenic. The medical evidence rested chiefly on the symptoms and post-mortem appearances, for no arsenic was discovered in the body. The mucous membrane of the stomach and intestines was found, throughout its whole extent, exceedingly inflamed and softened. The medical witnesses for the prosecution referred this condition to the action of arsenic; those for the defence considered that it might be owing to idiopathic gastro-enteritis, independently of the exhibition of any irritant. The circumstances of the case were very suspicious; but the prisoner was acquitted, not merely on account of the variance in the medical evidence, but from the absence of positive proof of poison, *i. e.* its detection by chemical analysis. This generally weighs much with a Court of law, although it is well known that arsenic cannot always be detected in the body of a person who has undoubtedly died from a large dose of this substance. It is right to state, as a warning to medical witnesses, that the judge who tried the case expressed regret that, on the non-discovery of poison in the contents of the stomach and intestines,—the soft parts of the body (the mucles) had not been examined according to the processes suggested by Orfila.—(See the published reports of the case by Mr. Holland and Mr. Dyson.)

In the healthy state, the mucous membrane of the stomach is pale and white, or nearly so, except during digestion, when it becomes reddened; and some observers have remarked that a slight redness has often remained in the stomachs of those who have died during the performance of the digestive process. When in contact with the spleen or liver, the stomach is apt to acquire a deep livid colour from the transudation of blood; and it is well known that the bowels acquire a somewhat similar colour from the gravitation of blood, which always takes place after death. None of these appearances are likely to be mistaken for the action of an irritant poison.

There is an important class of cases in which redness of the mucous membrane of the stomach is found after death, not dependent on the action of poison or on any assignable cause. These cases, owing to their being so little known, and involved in much obscurity, deserve great attention from the medical jurist;

since the appearances closely resemble those produced by irritant poison. A person may die without suffering from any symptoms of disordered stomach; but, on an inspection of the body, a general redness of the mucous membrane of this organ will be found, not distinguishable from the redness which is so commonly seen in arsenical poisoning. Several cases of this kind have occurred at Guy's Hospital; and drawings have been made of the appearance of the stomach, and are now preserved in the Museum collection. A record has been kept of four of these; and it is remarkable that, although in not one of them, before death, were any symptoms observed indicative of irritation or disease of the stomach, yet in all, the stomach was found more or less reddened, and in two extensively so. Such cases are not very common; but the certainty of their having occurred where poisoning could not be suspected, should place the witness on his guard, so that he be not led to countenance a suspicion of poisoning too hastily. In order to distinguish them, we must note whether there have been any symptoms during life, and their nature; as, in the above cases, there may have been no symptoms, or they may have amounted only to slight gastric disturbance. Under these circumstances, they could not be mistaken for symptoms of irritant poisoning. Such cases are only likely to lead into error, those who trust to this post-mortem appearance alone as evidence of poisoning; but no medical jurist, aware of his duty, could ever be so misled.

This redness of the stomach may truly occur where there is some ground for suspicion, as in the following case, communicated to the Medical Gazette by Mr. Tyson, of Beccles. A young woman, far advanced in pregnancy, died suddenly in a fit of syncope, soon after rising one morning. She had been in ill health previously; but there was nothing to indicate that she had taken poison: indeed, from what has been already said, the suddenness of her death was rather against the suspicion that she had died from such a cause. Yet after death, it was found among other appearances that the mucous membrane of the stomach was inflamed (reddened?) and thrown into rugæ. Although the case was very badly investigated by the coroner, who refused to allow a post-mortem examination, (which was made after the inquest only by consent of the friends,) it appears to me that this was an instance similar to those above described, the redness being due to some unknown cause, but not to poison. An interesting case, in which it is probable that this pseudo-morbid appearance of the mucous membrane was mistaken for the effects of irritant poison, will be found in the Ann. D'Hyg., 1835, i. 227. Dr. Yelloly long since remarked, that the mucous membrane of the stomach often presented a high degree of vascularity (redness) in cases of sudden death. He met with this appearance in the stomachs of some executed criminals, whose bodies were examined soon after they had undergone the sentence of the law. In a paper read before the Med.-Chir. Society in November 1835, this gentleman has re-examined the whole subject, and has given the results of numerous observations. They are of great interest to the medical jurist. Dr. Yelloly has endeavoured to show—1. That vascular fulness of the lining membrane of the stomach, whether florid or dark-coloured, is not a special mark of disease, because it is not inconsistent with a previous state of perfect health. 2. That those pathologists were deceived, who supposed, from the existence of this redness in the stomach, that gastritis sometimes existed without symptoms. 3. That erroneous conclusions as to the cause of death were frequently owing to the same mistaken observations:—the effects of putrefaction and spontaneous changes induced by the loss of vitality, being sometimes attributed to the action of poisons. 4. That the vascularity in question is entirely venous, the florid state of the vessels arising from the arterial character of the blood remaining in the veins for some time after its transmission from the arterial capillaries at the close of life:—the appearance is, however, sometimes due to transudation only. 5. That the fact of inflammation having existed previously to death, cannot be inferred merely from the aspect of the vessels in a dead part: there must at least have been symptoms during life. (See Medical Gazette, vol. xvii. p. 309.) Andral and other pathologists have adopted similar views, and these views have obviously a most important bearing upon medico-legal practice: since there is generally a tendency to suspect poisoning, whenever redness of the mucous membrane of the stomach is met with in the dead. Such a condition does not even prove the past existence of inflammation, unless there were symptoms during life or other marked effects of the inflammatory process in the alimentary canal. It can be no sign of poisoning, unless the presumption be supported by evidence from symptoms, or by the discovery of the poison: the absence of poison may, however, be sometimes explained by circumstances.

A case is mentioned by Foderé, where, in the body of a person who died suddenly, and poisoning was suspected, the œsophagus and stomach were found reddened. This was at first considered to be a confirmation of a suspicion, that the deceased had died from poison; but on inquiry, it was ascertained, that the redness was due to the colouring matter of a strong infusion of red poppies, which the deceased had been in the habit of taking. It is not likely that any person, moderately informed in his profession, would fall into such a mistake as this, the means of distinguishing all red colours by chemical tests being so simple; not to mention that mere washing might remove colours of this kind.

The redness of the stomach, in cases of poisoning, is so speedily altered by putrefaction, when circumstances are favourable to this process, as to render it impossible for a witness to speak with any certainty upon its cause. Putrefactive infiltration from the blood contained in the adjacent viscera and muscles, will give a reddish coloured appearance to a stomach otherwise in a healthy condition. Great dispute has arisen respecting the length of time during which redness of the stomach produced by an irritant will be recognizable and easily distinguishable from putrefactive changes. It is, perhaps, sufficient to say, that no certain rule can be laid down on the subject: it must be left to the knowledge and discretion of the witness. I have distinctly seen the well-marked appearances of inflammation produced by arsenic in the stomach and duodenum, in an exhumed body twenty-eight days after interment (*Reg.* v. *Gennings*, Berks Lent Ass. 1845;) and in another instance, referred to me by Mr. Lewis, the coroner for Essex, in August, 1846, the reddened state of the mucous membrane, in a case of arsenical poisoning, was plainly perceptible, on removing a layer of arsenic, *nineteen months* after interment. (See on this question, a case of suspected poisoning by Orfila, Annales D'Hyg. 1839, i. 127.) If, however, there be the least doubt respecting the origin of the discolouration, it would be unsafe to rely upon it, as evidence of poisoning. In the *Boughton* case, the medical witnesses stated that the stomach and viscera of the deceased were red, and presented the appearance of inflammation. In answer to a question put to him on the subject, the Crown witness, Dr. Rattray, said, that "*the post-mortem appearances confirmed his opinion of poisoning by laurel-water, so far as he might be allowed to form a judgment upon appearances so long after death.*" This very ambiguous answer led to the following cross-examination by the counsel for the prisoner.

C. "By your putting your answer in that way, do you or do you not mean to say that all judgment in such a case is unfounded?"

W. "I cannot say that, because from the analogy between the appearances in that body, and those distinguishable in animals killed by the poison I have just mentioned, I think them so much alike, that I am rather confirmed in my opinion with respect to the operation of the draught."

C. "Those bodies were *instantaneously* opened?"

W. "Yes; so much so, that there was the peristaltic motion of the bowels upon their being pricked."

C. "This" (the examination of the deceased's body) "was upon the *eleventh* day after Sir Theodosius's death?"

W. "Yes."—(Report of the trial of Donellan.)

Here it will be seen that the witness was comparing the appearances in the bodies of dogs *immediately* after they had been killed by poison, with those met with in the stomach of a person who had died *eleven* days before, and whose body had been buried and exhumed. Such a comparison was obviously wrong as a matter of medical evidence.

ULCERATION.—In irritant poisoning, the stomach is occasionally found ulcerated; but this is, comparatively speaking, a rare occurrence. In such cases the mucous membrane is removed in small distinct circular patches, under the edges of which the poison (arsenic) is often found lodged. Ulceration of the stomach is perhaps a more common result of disease, than of the action of poison. As a consequence of disease, it is very insidious, going on often for weeks together, without giving any indications of its existence, except perhaps slight gastric disturbance, with occasional nausea, vomiting, and loss of appetite. In this case the ulceration is commonly seen in small circumscribed patches. It is worthy of remark, as one means of diagnosis, that ulceration has never been known to take place from arsenic or any irritant poison, until *after* symptoms, indicative of irritant poisoning, have occurred. In ulceration from disease, the mucous membrane is commonly only reddened in the neighbourhood of the ulcer. In ulceration from poison, the redness is generally diffused over other parts of the stomach, as well as over the duodenum and small intestines. A case, however, occurred in Guy's Hospital, some years ago, in which, with a small circular patch of ulceration near the cardiac opening, the whole mucous membrane was red and injected:—but this singular condition of the stomach, so closely resembling the effects of an irritant poison, was unaccompanied by any marked symptoms during life. The history of a case previous to death, will thus commonly enable us to determine, to what cause the ulceration found, may be due. Care must be taken to distinguish ulceration from corrosion. Ulceration is a vital process; the substance of a part is removed by the absorbents as a simple result of inflammation. Corrosion, on the other hand, is a chemical action;—the parts are removed by the immediate contact of the poison: they are decomposed: their vitality is destroyed, and they combine with the corrosive matter itself. Ulceration requires time for its establishment, while corrosion is generally an instantaneous effect.

SOFTENING.—The coats of the stomach are not unfrequently found so soft, as to yield and break down under very slight pressure; and this may be the result either of poisoning, of some spontaneous morbid change in its structure during life, or of the solvent action of the gastric juice after death. As this change in the stomach, when caused by poison, is commonly produced by those substances only, which possess corrosive properties, it follows that in such cases, traces of their action will be perceived in the mouth, fauces, and œsophagus. In softening from disease, the change will be confined to the stomach alone, and it is commonly found only at the cardiac extremity of the organ. When softening is really caused by an irritant poison, it is generally attended by other striking and ambiguous marks of its operation. Softening is not to be regarded as a common character of poisoning: it is only an occasional appearance. I have met with a case, in which the coats of the stomach were considerably hardened by sulphuric acid. Softening can never be inferred to have proceeded from poison, unless other well-marked changes are present, or unless the poison be discovered in the softened parts. The stomachs of infants have been frequently found softened from natural causes:—such cases could not be mistaken for poisoning, since the history during life,—the want of other appearances indicative of poisoning, and the total absence of poison from the viscera, would prevent such a suspicion from being entertained.

Perforation.—The stomach may become perforated either as a result of poisoning or disease.

Perforation from poisoning.—This may occur in two ways:—1. By corrosion; 2. By ulceration. The perforation by *corrosion*, is by far the most common variety of perforation from poisoning. It is occasionally witnessed where the strong mineral acids have been taken, especially sulphuric acid:—the stomach, in such cases, is blackened and extensively destroyed,—the aperture is large, the edges are rough and irregular, and the coats become easily lacerated. The poison escapes into the abdomen, and may be readily detected by chemical analysis. The perforation from *ulceration*, caused by irritant poison (arsenic,) is but little known. There are, so far as I know, only three instances on record. In a great number of poisoned subjects examined during many years past at Guy's Hospital, not a single case has occurred. It must then be looked upon as a very rare appearance in cases of irritant poisoning.

Perforation from disease.—This is by no means an unusual occurrence. Many cases of this description will be found reported elsewhere. (Guy's Hosp. Rep. No. 8.) It is invariably fatal when it proceeds so far that the contents of the stomach escape into the abdomen; but sometimes the stomach becomes glued to the pancreas during the ulcerative process, and then the individual may recover. Several specimens of this kind of adhesion have been met with in post-mortem inspections. The symptoms from perforation commonly attack the individual suddenly, apparently while enjoying perfect health. Thus, then, these cases may be easily mistaken for those of irritant poisoning. The principal facts observed with regard to this formidable disease are the following:—1. It often attacks young females from eighteen to twenty-three years of age. 2. The preceding illness is extremely slight, sometimes there is merely loss of appetite or capricious appetite, with uneasiness after eating. 3. The attack commences with a sudden and most severe pain in the abdomen, generally soon after a meal. In irritant poisoning, the pain usually comes on gradually, and slowly increases in severity. 4. Vomiting, if it exist at all, is commonly slight, and is chiefly confined to what is swallowed. There is no purging:—the bowels are generally constipated. In irritant poisoning, the vomiting is usually severe, and diarrhœa seldom wanting. 5. The person dies commonly in from eighteen to thirty-six hours:—this is also the average period of death in the most common form of irritant poisoning, *i. e.* by arsenic;—but in no case yet recorded, has arsenic caused perforation of the stomach, within twenty-four hours; and it appears probable that a considerable time must elapse before such an effect could be produced by this or any irritant. In two cases of perforation of the stomach from disease in females, reported by Dr. Seymour to the Med.-Chir. Soc. November 1843, the one proved fatal in ten days, the other in a fortnight after the occurrence of the supposed perforation. The ulcers in the stomach were found to communicate with cysts. 6. In perforation from disease, the symptoms and death are clearly referable to peritonitis. 7. In the perforation from disease, the aperture is commonly of an oval or rounded form, about half an inch in diameter, situated in or near the lesser curvature of the stomach, and the edges are smooth. The outer margin of the aperture is often blackened, and the aperture itself is funnel-shaped from within outwards, *i. e.* the mucous coat is the most removed, and the outer or peritoneal coat, the least. The coats of the stomach, round the edge of the aperture, are usually thickened for some distance; and when cut, they have almost a cartilaginous hardness. These characters of the aperture will not only indicate, whether it be the result of poisoning or disease; but the absence of poison from the stomach, with the want of other characteristic marks of irritant poisoning, would enable us to say, that disease was the cause. Besides, the history of the case during life, would materially assist us in our diagnosis. The great risk in all these cases, is that the effects of disease may be mistaken for those of poisoning; for we are not likely to mistake a perforation caused by irritant poison for the result of disease. Among numerous instances, tending to show the medico-legal importance of this subject, I shall select one, which came before Mr. Hilton and myself for examination. A female in a noble family, aged twenty-three, died somewhat suddenly, under suspicious circumstances. She had been unwell for about three weeks, and was subject to occasional vomiting and disorder of the stomach. Still, her illness was so slight that it did not in the least interfere with the performance of her usual duties. One afternoon, about four o'clock, and about three hours after her last meal, she was suddenly seized with the most excruciating pain in the abdomen, and violent vomiting. Her skin was cold and clammy, and the abdomen tender and painful. It was suspected that she had taken poison; and magnesia and sulphate of magnesia were given to her. No poison was found in the room, and she strongly denied the imputation. The symptoms became worse, the vomiting more violent, and she died the following morning, about fifteen hours after her first seizure. On inspection all the organs were found healthy, except those of the abdomen. There were here strong marks of peritoneal inflammation: the intestines were loosely adherent to each other, and a quantity of lymph was effused around them. The cavity contained about a pint of liquid, which had escaped from an aperture in the stomach. This liquid was reserved for analysis. The stomach was laid open by making an incision along its greater curvature. It was empty. At the upper and posterior part, near the pyloric end of the smaller curvature, was an opening of an oval shape, about half an inch in its longest diameter. The edges were firm, hard, and smooth, presenting not the least appearance of laceration or ulceration. They were bevelled off from within outwards, being thinned towards the peritoneal coat, the aperture in which was much smaller than that in the mucous membrane. There was no sign of inflammation in the membranes around; but the peritoneum, about the edge of the aperture, had a black appearance, and the coats of the stomach were thickened. At the lower part, near the larger curvature, there were thick, irregular, black striæ, the mucous membrane being raised and blackened, but not softened. These striæ appeared like those produced by sulphuric acid; but there was no corrosion, and on applying test paper there was no acid reaction. The black matter was interspersed with a yellowish coloured substance. The liquids taken from the abdomen, as well as the coats of the stomach, were chemically examined; but not a trace of poison could be detected. Considering the time of the occurrence of symptoms, their nature, the absence of poison from the viscera and their contents,—the suspicion of poisoning was at once negatived, especially when the above facts were taken with the post-mortem appearances. The medical opinion given was, 1. That the deceased had died from peritonitis, caused by extravasation of the contents of the stomach. 2. That this extravasation was owing to a perforation of the coats of the organ, caused by slow and insidious disease, and not by poison.

It has been hitherto supposed that perforation of the stomach must necessarily prove fatal. This is undoubtedly the ordinary result, but the fatal effect depends on peritonitis, excited by extravasation of the contents of the organ. Under favourable circumstances, and by judicious treatment, no extravasation may take place, and the individual may entirely recover;—as the aperture ultimately becomes closed by adhesion to the surrounding viscera. Of this closure of ulcerated apertures in the stomach, several specimens are preserved in the museum of Guy's Hospital. This fact is interesting to the medical jurist, as a case might, from the symptoms, be mistaken for one of poisoning; although, when taken on the whole, they are unlike those produced by irritant poison. An apparently well marked instance of recovery from perforation is reported by Dr. Hughes. (G. H. Rep. N. S. iv. 332.) The patient recovered from the first attack, but ultimately died from another perforating ulcer, which led to extravasation. (Case by Mr. Hilton, ib. 343.)

Spontaneous or Gelatinized Perforation.—The stomach is occasionally subject to a spontaneous change, by which its coats become softened and give way, generally at the cardiac extremity. As the extravasation of the contents of the organ in such a case never gives rise to peritoneal inflammation, and no symptoms occur prior to death to indicate the existence of so extensive a destruction of parts, it is presumed to be a post-mortem change, and the stomach is supposed to undergo a process of solution soon after death. It is commonly attributed to the solvent action of the gastric juice, the spleen, diaphragm, and other viscera being sometimes softened. (For some remarks on this subject, by Dr. Budd, see Med. Gaz. xxxix. 895.) In January 1845, I met with an instance of this perforation in a child between two and three years of age. It was seized with convulsions, became insensible, and died twenty-three hours afterwards. After death, the cardiac end of the stomach was found destroyed to the extent of three inches; and the edges were softened and blackened. There was no food in the stomach, nor had any thing passed into the organ for thirty-two hours before death! It was therefore impossible to ascribe death to the perforation, or the perforation to poison. (For a full account of this case, see Med. Gaz. xxxvi. 32.) In October 1846, I found the same condition of the stomach in an infant aged nineteen months, suspected to have died from poison administered to it three months before. The cause of death in this case was mesenteric disease. The stomachs of young children at the cardiac end are always very thin, and thus but a slight softening action is required to bring about a destruction of the coats. The same effect is observed in rabbits, in which the coats of the stomach are remarkably thin, and are often found quite pulpy on inspection, irrespective of disease or the presence of poison. This form of perforation is, so far as I can judge, by no means common. It is reported to have been met with in children affected with hydrocephalus,—in those who have died from typhus fever;—and, according to Andral, in females who have died during parturition. Dr. Macintyre informed me that he had met with two cases of this kind of perforation in young subjects affected with diabetes. The conditions for its production, whether local or constitutional, and the circumstances under which it occurs, are very obscure. (Med. Gaz. xxxix. 897.) The fact of most importance to the medical jurist is, however, that it is unattended by any marked symptoms during life. Some French pathologists describe cases of what they term *gelatinized* perforation, in which gastric disorder had existed. Chaussier, indeed, believed that this form of perforation always depended on a particular disease of the organ; and he denied, from the results of his own experiments, that the gastric juice had any solvent action. (Flandin, Traité des Poisons, i. 259.) The inspection of the body, with the general history of the case, must, however, suffice to remove any difficulty in forming an opinion as to whether the extensive destruction commonly met with, has or has not arisen from poison. Thus, the aperture, which is always situated in that part of the stomach which lies to the left of the cardia, is very large, of an irregular form, and ragged and pulpy at the edges. These have the appearance of being scraped; the mucous membrane of the stomach is not found inflamed. There is occasionally slight redness, with dark brown or almost black striæ in and near the dissolved coats, which have an acid reaction. It can only be confounded with perforation by the action of corrosives; but the well-marked symptoms during life, and the detection of the poison after death, together with the changes in the fauces and œsophagus, will at once indicate the perforation produced by corrosive poison. The only case in which any mistake is likely to occur, is where, conjoined with the discovery of perforation after death, there may have existed some symptoms of irritation in the alimentary canal during life. It is possible that a person may die under symptoms somewhat resembling irritant poisoning, and after death the gastric secretion may destroy the parietes of the stomach; but such a singular combination of circumstances must be most unusual. This, however, signifies little in a legal point of view, for persons charged with the crime of poisoning, are frequently acquitted on the barest medical possibilities. One case of this doubtful character is on record. I allude to that of *Miss Burns*, for the murder of whom, by poison, a Mr. Angus of Liverpool was tried in the year 1808. It is not necessary to enter into the particulars of the case; since the post-mortem appearances are imperfectly described in the report. Although the symptoms, resembling irritant poisoning, under which the deceased laboured, were not accounted for, yet there was great reason to believe that they were not connected with the perforation of the stomach, which, on the whole, bore the characters assigned to that produced by the gastric secretion. The charge of poisoning was not sustained by chemical or pathological evidence, and the prisoner was acquitted. The evidence given on this trial, is well worthy of the attention of every medical practitioner. It shows on what a nice balance of proofs charges of poisoning sometimes rest, and how important it is that a medical jurist should be acquainted with all the circumstances under which perforations of the stomach may occur.

Perforation of the Œsophagus and Intestines.—Other parts of the alimentary canal are liable to become perforated: but not, so far as I have been able to ascertain, by the action of poison. The œsophagus may become softened by the contact of corrosive poison, but this rapidly passes through the tube and lodges in the stomach. As Dr. Christison observes, it is not probable that a corrosive poison could ever perforate the intestines from within outwards, since its action would be chiefly expended on the stomach, and it is not likely to reach any portion of the intestines in a state sufficiently concentrated to destroy the coats by chemical action. (On poisons, 149.) If a large quantity of corrosive poison flowed through an aperture in the stomach upon the intestines, then the coats might become destroyed from without inwards. The lower portion of the œsophagus, and various parts of the intestinal tube, have been found in several instances softened and destroyed, the aperture presenting all those characters which have been described in speaking of spontaneous or gelatinized perforation of the stomach. This change in the œsophagus is ascribed to a solvent action of the gastric juice, which enters the tube by regurgitation; but this explanation cannot apply to the intestines. An interesting case of softening of the intestines, from the duodenum to the sigmoid flexure of the colon, is reported by Mr. J. Smith: the child died of hydrocephalus. (See Med. Gaz. ii. 619.) The intestinal tube may become perforated in any part by ulceration, depending either on disease or on the action of irritant poison; but ulceration of the intestines from poison proceeding to perforation of the coats, is a very rare condition.

Cases of *perforation of the intestines from disease* are occasionally met with. They require the attention of the medical jurist, since they may be easily mistaken for cases of poisoning. The following instance of perforation of the duodenum is reported by Mr. Bailey (Med. Times, Dec. 19, 1846, p. 223.) A female, æt. 28, was taken suddenly ill. There was great anxiety of countenance, small and frequent pulse, cold extremities, with occasional eructations of wind and vomiting. She suffered excruciating pain in the region of the duodenum, which caused her to double up her body. Her bowels had been only partially opened the day before. In about nine hours there was great tenderness of the abdomen; the pulse was smaller; but the bowels, in spite of the administration of medicine, were still unmoved. She gradually sank, and died in about fourteen hours and a half from the time she was first seized. Thus far the symptoms would indicate that the case was one of acute peritonitis, and not of irritant poisoning. Nevertheless the circumstances were extraordinary, and rumours were spread that her husband had poisoned her. The deceased, it appeared, had only been married the previous day; she and her husband did not retire to rest until three o'clock in the morning, and the attack came on suddenly, by a fit of severe pain, at nine o'clock, *i. e.* six hours after they had retired to rest. An inspection of the body showed all the usual marks of peritoneal inflammation, and the duodenum, in its transverse portion, was found to have a circular opening in it, surrounded by a blank margin externally; while internally the perforation seemed larger, its sides sloping off. The mucous membrane was softened for some distance around the ulceration, affording evidence of the existence of previous inflammation. It appeared that the deceased had been a cook up to the day of her marriage. The only symptom manifested before the attack was, that she had been for some time subject to pain in her right side. (See also Cormack's Ed. Jour. June 1845, p. 445; and Lancet, July 18, 1846, p. 67.)

An interesting case of perforation of the ilium, in which two apertures were found, is reported by Dr. Zartmann. (Casper's Wochenschrift, März 14, 1846.) Death took place in two days, from peritonitis. One aperture was only the sixth of an inch in diameter. The edges of both were smooth, as if punched out.

There is another insidious form in which perforation of the intestines may present itself, and cause fatal peritonitis. This is by the formation of an ulcerated aperture in the appendix vermiformis cœci, of which two cases have been communicated to me by a former pupil, both occurring in young men. The perforation was produced in these instances by the pressure of a hard substance lodged in the extreme end. In one instance I analysed this hard concretion, and found it to consist of inspissated mucus, biliary matter, and a large quantity of carbonate of lime. It was of an irregular form and structure, and about the size of a large pea. In both instances death was caused by peritonitis, produced by extravasation of the contents of the intestines, and the aperture was so small that it might have been easily overlooked. It is remarkable that the introduction into the appendix cœci of any foreign substance, as the pip of an apple, or a small bean, or cherry stone, is liable to excite fatal inflammation, ulceration, or gangrene, and death. An interesting case of this kind has been reported by Mr. Nelson (Amer. Journ. Med. Science, Feb. 1847, p. 258.) The fact is important in relation to the causes of sudden death.

Perforation by worms.—It is now generally admitted that the various species of worms which infest the alimentary canal of the human subject, may, in some rare cases, by irritating or even perforating the intestines, give rise to symptoms which may be mistaken for those of poisoning. One of the most recent writers on toxicology, M. Flandin, denies that perforation can ever take place from this cause (Des Poisons, i. 304, 1846;) but as this denial is based on theory, while the statement which he impugns rests upon good authority, it is unnecessary to discuss this as an unsettled question. He supports his opinion by the authority of Rudolphi, who states that the entozoa have no organs capable of perforating the intestinal coats,—by the view entertained by M. Dujardin, that worms are never injurious to animals, because they are often found in large numbers when the previous health of the animal has been uninjured,—and by the statement of Andral, who, however, merely says, that if worms have the power of perforating the intestines, the cases in which this happens, are exceedingly rare—a conclusion in which most pathologists will entirely agree with him. Several instances are on record in which perforation has been thus caused, and the worm or worms have been found in the peritoneal cavity. A well-marked case of perforation of the ileum by an ascaris lumbricoides, leading to death from effusion and peritonitis, is reported by Mr. Kell. The opening through the intestine was of a circular form, and corresponded in magnitude with the size of the worm, which was eight inches long. The worm was found among the intestines, between the umbilicus and pubes. (Med. Gaz. ii. 650.) These worms may even perforate the abdominal parietes. (See cases by Dr. Young, Med. Gaz. ii. 748.) Vogel says of ascaris lumbricoides, that in certain cases it is capable of perforating the intestine, by thrusting asunder with its head the fibres of the intestinal coats. (Pathological Anatomy, Day's translation, p. 464.) When perforation takes place from this cause, it will be indicated by the discovery of the entozoon in the cavity of the peritoneum, or near the aperture in the intestine. (See a case in Cormack's Ed. Journ. June 1845, p. 447.)

In a case of suspected irritant poisoning, when worms are discovered in the intestinal canal, an attempt is not unfrequently made in the defence to refer the symptoms of irritation to the presence of these entozoa. If the symptoms of poisoning be well marked, and some of the poison be discovered in the body, such a defence must be a hopeless struggle against medical facts. This objection was taken to the evidence from symptoms in one case of arsenical poisoning; but the disturbance occasioned by worms is commonly slight, and is very rarely accompanied by vomiting and purging. A solitary instance is quoted by Dr. Christison, in which a child appears to have died under symptoms of narcotico-irritant poisoning as an effect of worms. Several hundred ascarides were found in the intestines, and thirteen in the stomach. (On Poisons, 133.) In two instances of arsenical poisoning which have lately occurred, I found a large ascaris lumbricoides in the small intestines. There could not, however, be the slightest doubt that the sole cause of death was poison. In one instance the ascaris was well washed from adhering mucus, and examined for arsenic by Reinsch's process:—the poison was abundantly discovered in it. (G. H. Rep. N. S. iv. 462.) Under such circumstances it would be impossible to refer the symptoms of irritation to worms. A case will be mentioned in the chapter on MIXED MINERAL ACIDS, in which, on examining the body of a person who had taken an ounce and a half of muriatic acid, the jejunum was found perforated by a lumbricus; but the acid was beyond all doubt the cause of death.

That, however, the presence of worms in the body may become a ground of defence, is sufficiently proved by two cases reported by M. Flandin. A young man was charged with having poisoned his father with arsenic. A very small quantity of the poison was detected in the tissues of the body only. The report of the inspection stated, that there was a large number of the ascarides lumbricoides in the intestinal canal, and some were even found in the œsophagus. A discussion on the cause of death arose between the counsel employed in the defence, who based his arguments on a memoir by M. Raspail, and the medical witness, who had discovered arsenic in the body, but especially in the liver. It was contended that the quantity of arsenic found was not sufficient to cause death (for the fallacy of an argument of this kind, see post CHEMICAL ANALYSIS, p. 136;) and that the presence of the ascarides explained satisfactorily the cause of the symptoms, and their rapidly fatal course. The deceased, it was alleged, had been suffocated by the worms, which had caused compression of the bowels, and had even ascended into the œsophagus! The witnesses replied, that arsenic was never found in the healthy human body,—that the presence of worms could not account for the presence of arsenic in the tissues,—while the detection of the poison, on the other hand, accounted for all the symptoms, even for the passage of the worms into the œsophagus as a result of violent vomiting. To no other circumstance could death be attributed.

In the second case, an empiric applied to the cancerous breast of a female, a plaster covered with arsenious acid. Symptoms of poisoning by absorption followed, and the woman died. Arsenic was detected in the liver. An ascaris was found in the intestines. On removing the whole of the viscera, which were in a highly putrefied state, a portion gave way, and the worm appeared through the lacerated aperture. There had been no extravasation, nor any mark of peritonitis. The defence was, that the deceased had died from perforation of the intestine by the worm. This was denied by the witnesses, who contended that had this been the case, there would have been extravasation. The escape of the worm was owing to accidental laceration of the viscera during the inspection. The symptoms of the disease were clearly those of poisoning and not of peritonitis. (Des Poisons, i. 307 and 507.)

Five species of parasitic worms have been hitherto found in the human intestines:—1. *Ascaris lumbricoides*, or round worm. 2. *Trichocephalus dispar*, or long thread worm. 3. *Oxuris*, or *Ascaris vermicularis*, common thread worm. 4. *Tænia solium*, common tape worm: and 5. *Tænia lata* (*Bothriocephalus*) broad tape worm.

1. The *Ascaris lumbricoides* is very common. It is a round cylindrical worm pointed at both ends, especially at the anterior extremity: it varies in length from one inch to ten or even fifteen inches. It is usually of a whitish or brownish red colour, and occasionally blood-red. A delicate furrow runs along the body on both sides. This worm occurs especially in young children, and is often found in great numbers without any disturbance of health. When accumulated in large quantities, the ascarides are liable to excite intestinal irritation.

2. The *Trichocephalus dispar* is a thin and thread-like worm, varying in length from an inch and a half to two inches. It is usually white, sometimes slightly coloured: it frequently occurs in the large intestines, especially in the cœcum. According to Vogel, it is found, gregarious or solitary, in nearly half the subjects examined;—it is firmly adherent by its capillary head to the mucous membrane. Those persons, in whose bodies this worm has been found in very large numbers, have not exhibited any symptoms of its presence.

3. *Oxyuris.*—This is a thin white worm, smaller than the trichocephalus; it occurs in the large intestines, particularly in the rectum, and it is especially common in infants. The male, which is smaller than the female, varies in length from a line to a line and a half. It is spirally coiled at the tail, and often completely assumes the form of a ring.

4. *Tænia solium.*—A riband-shaped, long pointed worm, of a milk-white or yellowish colour. Its length sometimes exceeds twenty feet; its breadth varies from a quarter to a half an inch, but it is less at the head; its greatest thickness does not exceed the twelfth of an inch. It inhabits the small intestines of the human subject, but only in certain parts of the world. It remains in the body for a long period, without its presence being indicated by the slightest symptoms.

Tænia lata.—This worm closely resembles the preceding. It varies in length from one foot to twenty or even forty feet. There are but very slight differences between this and the tænia solium or vulgaris. (See on this subject Vogel's Pathological Anatomy, Day's Translation, p. 470.)

CHAPTER X.

ON THE EVIDENCE OF POISONING FROM CHEMICAL ANALYSIS—NOT ESSENTIAL—RULES FOR CONDUCTING AN ANALYSIS—CIRCUMSTANCES UNDER WHICH AN ANALYSIS MAY BE REQUIRED—FAILURE OF CHEMICAL EVIDENCE—CAUSES OF THE NON-DETECTION OF POISON—LOSS BY ELIMINATION AND PUTREFACTION—EVIDENCE FROM THE QUANTITY FOUND IN THE BODY—DELICACY OF ANALYSIS—POISONS IN TESTS AND APPARATUS—DANGER OF PREMATURE OPINIONS—FALLACIES IN CHEMICAL ANALYSIS.

Convictions without chemical evidence.—It has been supposed that chemical evidence of poisoning was always necessary, and that the *corpus delicti* was not made out, unless the poison were discovered by a chemical analysis. This, however, is not a correct view of the matter. There are many poisons which cannot, at present, be detected by chemical analysis, and among those susceptible of analysis, there are numerous circumstances which may occur to prevent their detection in the food, the vomited matters, or the contents of the viscera, in the dead. If such a rule were recognised by law, many criminals would escape conviction. All that is required legally, is that there should be satisfactory proof of a person having died from poison;—the discovery of poison in the body, is not necessarily evidence of its having caused death, nor is its non-discovery evidence that death has not been caused by it. If by the symptoms and post-mortem appearances, with or without moral circumstances, it can be made clear to the minds of a jury that death has been caused by poison, nothing more is required; the evidence from chemical analysis may be then safely dispensed with. In cases of murder, the law commonly requires that the body of a deceased person should be produced, in order that the cause of death may be verified; but this is not absolutely necessary, for several convictions for murder have very properly taken place, where the bodies of the murdered persons have not been forthcoming. Thus, then, we must not suppose that a charge of poisoning cannot be sustained without chemical evidence being produced of the nature of the substance taken. The fact of a poison having been used, as well as its nature, may be determined from other circumstances. In the case of *Donellan* already referred to, the only evidence of the nature of the poison used, was the odour perceived by a non-professional person. The effects which followed made up for the want of clear chemical proof of its nature. As some objections have been offered to the propriety of a conviction in this case, I may refer to two others:—one the case of a man named *Thom*, tried at the Aberdeen Autumn Circuit, 1821, for poisoning a person named Mitchell with arsenic. No trace of poison could be detected; but a conviction very properly took place on evidence from symptoms and post-mortem appearances, coupled with moral circumstances. A still more recent instance occurred at the Monaghan Lent Assizes, 1841, where a woman was convicted of poisoning her husband, although the nature of the poison could not be determined by the most carefully conducted chemical analysis. The poison was considered to have been aconite. (See also *Humphrey's* case, ante, p. 47.)

On the other hand, when the other branches of evidence are weak or defective, the detection of the poison by chemical analysis becomes of such importance, that if it fail, an acquittal will follow. Conjoined with strong moral circumstances, chemical evidence will often lead to conviction when post-mortem appearances are entirely wanting, and the evidence from symptoms is very imperfect. The great value of chemical evidence in otherwise doubtful cases of poisoning, was never more strongly shown than at the trial of *Mary Ann Burdock* at Bristol, in 1835, for poisoning Clara Smith with orpiment. The body of the deceased had been buried fourteen months: it was exhumed, and the poison discovered in the stomach and viscera. It must be clear to all medical jurists, that had it not been for the detection of the poison in the viscera after this long period of time, the prisoner would most probably have been acquitted. We cannot therefore be surprised to find that it is this branch of evidence which is deemed most satisfactory to the public mind, and which is earnestly sought for by our law authorities on charges of poisoning. The reason is, that in most cases, it demonstrates at once the means of death; while symptoms and post-mortem appearances are, as we have seen, fallible criteria, unless many circumstances, often difficult of appreciation, are fully considered by the medical witness. Many coroners are not sufficiently aware of the importance of this branch of evidence in cases of suspicious death. In several instances of recent occurrence, the fact of poisoning has been established by a chemical analysis of the contents of the stomach long after interment, although verdicts of natural death had been previously returned. In order to put a check on the extensive but secret destruction of life by poison in this country, chemical analysis of the contents of the stomach should be more frequently made. Impunity in one instance, as a result of a careless inquiry, uniformly leads to the perpetration of a series of murders.

Rules for conducting an analysis.—Before proceeding to the analysis of any suspected substance, we should, if possible, make ourselves fully acquainted either with the symptoms or post-mortem appearances, or both, observed in the person suspected to have been poisoned. We may by a knowledge of these facts determine, à priori, whether we shall have to search for a narcotic, irritant, or corrosive substance. The kind of poison may often be predicted from the symptoms and post-mortem appearances, and our analysis directed accordingly. I have, however, known more than one instance, where an irritant poison has been sought for in the contents of the stomach, when every fact connected with the death of the party, as well as the rapidity with which death took place, tended clearly to show that if any poison had been used, it must have been one of the pure narcotics. It is not unusual to find the examination of medical witnesses misconducted in Courts of law, in relation to the effects of poisons. The deceased may have died from a narcotic, while questions relative to the action of irritants alone, will be put by the counsel for the prosecution and defence. The chemical evidence may be divided into several branches. The analysis may extend—

1. To the pure poison. We may be required to state the nature of a substance (part of the poison administered) found in the possession of a prisoner.

2. The analysis may be confined to a portion of the substance of which the affected party partook; and here the poison is usually mixed up with liquids or solids of an organic nature. The steps of the analysis become then rather more difficult. *a.* There may have been various substances combined in a meal, and the poison have been mixed with one substance only. This will show the necessity for examining separately the various articles used at a meal, if we wish to discover the real vehicle of the poison. *b.* Symptoms of poisoning may occur after the eating of a pudding. A part of the pudding may be analysed, and no poison discovered; because the poison, instead of being incorporated with the dough, may have been loosely sprinkled like flour over the exterior only. *c.* A similar circumstance may occur in the poisoning of a dish of meat. The gravy may be poisoned, and not the meat. A case of this kind occurred to Dr. Christison. A whole family was attacked with symptoms of poisoning after a meal on roast beef. The meat was examined, but no poison could be discovered. It was then ascertained that the poison had been mixed with the gravy,

and those who had taken the meat without the gravy suffered but slightly. In one instance, which occurred lately, arsenic was placed instead of salt on the edge of the plate of the deceased. (The *Queen* v. *Jennings*, Berks Lent Assizes, 1845.) No other person experienced symptoms of poisoning after the meal, except the child who ate out of that plate. In the case of *Bodle*, tried in 1833, the deceased was proved to have been poisoned by arsenic administered in coffee. The coffee was kept ground in a bottle, to which every one of the family had access; and there could be but little doubt, from the circumstantial evidence, that the poison had been mixed with the coffee in this bottle. That which remained in the bottle was carefully examined by the late Mr. Marsh, but no trace of arsenic could be detected. The poison had most probably been mixed with the *upper stratum* only of the powered coffee, and the whole of the poisoned portion had been used for breakfast. A remarkable fact was brought out in the case of the *Queen* v. *Edwards*, (Central Criminal Court, November, 1844.) The deceased, it was stated, had died from drinking part of the contents of a bottle suspected to contain sugar of lead,—but it was proved that some of the same liquid had been drunk by another person the night previously without any injury resulting. The medical witness explained this by saying that the poison existed as a crust in the bottle, which might have been detached in one case and adherent in the other. A somewhat similar case is given under the section on CARBONATE OF LEAD. (See that compound, post.) Facts of this kind are of some medico-legal importance: they will often enable a witness to explain certain anomalies in cases of poisoning. By bearing them in mind, it is easy to understand, how it is that one or two persons only will suffer at a meal made in common or on the same article of food, while others will escape.

3. The chemical analysis may be directed to the matters *vomited* and evacuated. In irritant poisoning, a large quantity of poison is often expelled in this way, and may be detected especially in the matter first vomited. In a suspected case an immediate analysis should be made of the matters ejected from the stomach. They may be regarded as furnishing to the medical jurist, the proofs required to establish the corpus delicti.

4. If death has ensued, an analysis of the contents of the stomach and intestines must be made. Supposing no vomiting to have occurred, or that this has been slight, and death has taken place speedily, then we may expect to find abundant traces of the poison in the viscera. If no poison should be found in the stomach, the contents of the duodenum and the other small intestines, as well as of the rectum, must be separately examined. If the poison be of a mineral nature, and cannot be detected in the contents, it must be sought for in the tissues of the viscera, especially of the liver and spleen.

It is obvious that one or several of these sources of chemical evidence may be wanting, and it is rare in any one case of criminal poisoning that all are open to the medical witness. The detection of the poison in the vomited matters during life, and in the viscera after death, is of course the most satisfactory kind of chemical evidence; since, cæteris paribus, it is a clear proof of poison having really been taken. It is difficult to admit the supposition that it should have been designedly introduced after death; besides, in such a case, the absence of all marks of vital reaction, and of any symptoms during life indicative of poisoning, would remove such a suspicion. If the poison be detected in the tissues of any of the organs, there can be no doubt of its having been introduced into the body during life. The presence of poison in the viscera or their contents, with such marks of vital reaction as are known to be produced by the particular substance, as for instance, inflammation in the case of the irritants, affords the strongest presumptive evidence of death from poison, open to be rebutted by other proofs of death from disease, under which the deceased might have been labouring at the time.

Causes of the non-detection of poison.—But let us take the case, that chemical evidence is entirely wanting, and that no poison is detected under any of the circumstances mentioned: if there be other facts to render death from poisoning probable, we must endeavour to explain why this important branch of evidence has failed. There are few medical jurists who have not met with cases where, although undoubtedly death was occasioned by poison, whether irritant or narcotic, not a trace of the substance could be detected in the solids or liquids of the body. The non-discovery of poisons in cases of poisoning may depend

1. *On the nature of the poison.*—In the present state of our knowledge, chemistry, with few exceptions, furnishes us with the means of identifying with certainty a mineral poison only. The greater number of vegetable poisons are beyond the reach of chemical analysis. Botanical characters may sometimes serve to point out the nature of the substance; but only in those instances where the plant has been swallowed in the state of leaves, roots, or seeds. If the extract or inspissated juice has been administered, or if the poison were in the form of infusion, tincture, or decoction, a chemical analysis will commonly be of no avail. The same remarks apply to the powerful alkaloids extracted from vegetables. It is true, that there are tests for morphia, strychnia, and a few others; but these are on the whole unsatisfactory as a basis for chemical evidence of poisoning. Again, poisons which are of a highly *volatile* nature, may be speedily dissipated; so that in a few hours or a few days after death, none may be discovered. Alcohol is well known to pass away so rapidly, that no spirituous odour may be perceived in the contents of the stomach, although the individual may have died speedily, and the body be inspected within six or eight hours after death. Prussic acid may be in like manner rapidly dissipated. (See Prussic Acid.)

2. *Influence of vomiting and purging.*—The non-detection of poison in the viscera, may be owing to its having been expelled by the excessive *vomiting* and *purging*. In all such cases, however, the poisonous substance ought to be found in the vomited matters. In two instances of poisoning by sulpuric acid, —in two of arsenic, and in one of oxalic acid, although death took place with the usual rapidity, I could not detect any of the respective poisons in the stomachs of the deceased. Similar cases are to be found in most works on medical jurisprudence. It may, however, be fairly inferred that in all cases of irritant poisoning, where the vomiting and purging have been slight, some portion of the poison ought to be found in the body, if the individual have died within the average period, *i. e.* if he have not survived more than two or three days. Should none be present under these circumstances, it may be a question whether death was really due to poison. It is not at all probable that a common dose of arsenic would be entirely removed by absorption in the course of two or three days. (See the case of *Queen* v. *Hunter*, Liverpool Lent Assizes, 1834, ante, p. 30.)

3. *Loss by absorption and elimination.*—Solid poisons are usually detected without difficulty, because they are generally administered criminally in very large doses; but in cases of chronic poisoning, *i. e.* where the substance is administered in small doses at long intervals, chemical analysis will sometimes fail: for the poison may become entirely absorbed and eliminated. The researches of Orfila and others have proved that most metallic irritants enter into the circulation, and become diffused over the body, even to the extremities of the fingers and toes. Orfila has discovered that arsenic is especially liable to be excreted with the urine; it has also been detected in the serous exhalation of the pleura. Thus, if the dose of arsenic be small, or the poison be taken in a state favourable for absorption, *i. e.* in solution, no trace of the substance may be found in the body, unless death takes place very speedily.

It cannot be denied, that the great facility with which chemical analysis is applied to the detection of most irritant poisons, is due to the ignorance of those who criminally administer them. A mineral poison is commonly given in the form of a loose powder, undissolved; and it is then easily susceptible of analysis. Instances of extraordinary depravity have, however, occurred, in which persons have shown themselves to be acquainted with these facts, and they have endeavoured so to destroy their victims, as to frustrate the usual means of detection. A case was tried at Mayence in March, 1835, in which the evidence clearly proved, that the prisoners had poisoned the deceased and several persons previously, by administering to them arsenic in a saturated solution in water. One of them confessed that she had boiled the poison in water, allowed it to cool, filtered the solution, and then administered it by small quantities at a time in wine, milk, gruel, and other liquids. On one of these occasions, the dose of poison happened to be so large, that it operated fatally,—a circumstance which led to the detection of the crime. As might have been anticipated, not a trace of arsenic could be discovered on analyzing the contents of the viscera of those who had perished in this manner. A case somewhat similar is related by Dr. Christison, as having occurred in Scotland (p. 319.) The celebrated Acqua Toffana appears to have been a pure solution of arsenic.

I have elsewhere (ante, p. 30) adverted to the case of *Hunter*. The time required for the expulsion of arsenic from the body was here material. The deceased survived three days, and had suffered during the whole of the time from vomiting and purging. One of the medical witnesses stated that the arsenic might have been carried off by insensible perspiration and other means in progress for its elimination, in the course of two or three days. The Judge then put the question, would all traces of arsenic be likely to be carried away if the poison were taken at six o'clock on Friday night, and the person died on Monday evening? *Witness:* All from the stomach and bowels. The *Judge:* But from the flesh? *Witness:* I cannot tell. The state in which the arsenic was supposed to have been given was not in evidence. It was alleged to have been administered in milk, which the counsel thought might act as an antidote to it; but this would not effect the poison chemically, or interfere with its absorption. It is certainly quite possible that violent vomiting and purging at intervals, for less than three days, may carry off the poison from the contents of the viscera. I have met with several instances in support of this rapid expulsion of large doses of arsenic (G. H. Rep. April 1837, p. 79;) but it is almost equally certain, that, had an analysis been here made, the absorbed arsenic would have been discovered in the tissues of the viscera, supposing it to have been administered and to have caused death. The exact time which it requires for arsenic to be entirely removed from the body by elimination after its absorption, is undetermined. I have elsewhere stated, that the French medical jurists assign a period of ten, twelve, or fifteen days (survivorship) for its entire disappearance from the body, when taken in an ordinary dose (see p. 30.) Alcohol and prussic acid may also entirely disappear by absorption, and by elimination through the lungs. An instance has lately been communicated to me where a man by mistake swallowed forty-five drops of prussic acid of two per cent. He lay insensible for four hours: he then vomited and recovered. The vomited matters had not the least odour of the poison.

4. *Influence of treatment.*—Some poisons may be removed by *treatment*, as under the administration of antidotes and the use of the stomach-pump. Others appear to undergo a change analogous to digestion from the action of the secretions of the stomach upon them. These are chiefly poisons belonging to the organic kingdom; and this has been suggested by Dr. Christison as a reason why, in many cases of rapid poisoning by opium, no trace of the poison has been discovered.

5. *Loss by putrefaction.*—Although poisons may remain in the body at

the time of death, still they are liable to disappear during the progress of the putrefactive process. Soluble poisons contained in the viscera are soon lost. I have found in the stomach no trace of a large dose of oxalic acid given to an animal which had been buried twenty-eight days. Organic poisons easily undergo decomposition: mineral poisons resist these changes. Thus arsenic, it is well known, may be found after many years. In two cases lately, I detected this poison in the coats of the stomach nearly two years after the burial of the bodies. White arsenic, then becomes yellow by its conversion to sulphuret, and forms a very deep yellow stain in the coats of the organ. How long absorbed arsenic remains in the soft organs it is not easy to say; and the result of an analysis must depend materially upon the quantity which happens to be retained by the organs at the time of death. If this should have been small, there can be but little hope of detecting it at a long period after interment.

OBJECTS OF A CHEMICAL ANALYSIS.—A chemical analysis is commonly directed in toxicology to the determination of two points;—1. Of the *nature* of the poison. 2. Of the proportion, or *quantity*, in which it has been taken.

1. The *nature* of the poison and the probable quantity administered, are usually stated in the indictment; but it is not absolutely necessary for conviction that the substance thus stated, should be proved to have been that which was actually administered. The purposes of the law are considered to be fulfilled if the kind of death be substantially proved:—thus it is only necessary to prove that the person was poisoned. A man may be indicted for administering corrosive sublimate; but the medical evidence may show that the poison was in reality arsenic or prussic acid;—still the prisoner may be convicted of the crime, the variance in the means alleged being immaterial. This is, in many respects, fortunate; since a person may be convicted in spite of any imperfections existing in the original analysis.

2. The *quantity* of poison administered is generally stated conjecturally; but it is sometimes in the power of a witness to give a tolerably accurate statement of the quantity taken, when any portion of the original vehicle of the poison is discovered. Thus, all solid substances given for analysis should be first weighed;—and all liquids measured: a quantitative analysis may then be performed at any subsequent period. The chief question in law in regard to the quantity of poison is:—whether it was sufficient to destroy life, or to produce any serious effects? Thus, the malicious intention of a prisoner is often to be inferred from the quantity of poison existing in the substance administered. A case occurred some years since, in which a man was capitally indicted for administering oxalic acid with intent to murder. The poison was introduced into coffee, served for the prosecutor's breakfast. There could be no doubt of its presence; but on estimating the quantity, Mr. Barry discovered that it was only in proportion of about ten grains to a pint, a quantity which he considered insufficient to produce any serious effects on the body. The prisoner was acquitted; but it is obvious, that had the proportion been an ounce to a pint, the malice of his act would have been apparent. This case shows that a medical jurist must not be content with merely determining the presence of poison in suspected liquids,—he should also determine the quantity. The law presumes upon the innocence rather than upon the guilt of an accused party, when the evidence fails in showing, from the small quantity of the poison administered, that the act was malicious. If a man gave to another a few drops of sulphuric acid in a large quantity of water, we should not infer that his intention was to murder; but if he administered a large quantity of the acid in an undiluted state, the malice of the act would be at once apparent. Presumptions of this kind must, of course, be affected, as well by the nature of the poison, as by the moral circumstances adduced in evidence. A prisoner has sometimes alleged in his defence, that he did not know the substance to be a poison, and that he did not administer it with intent to kill. The law, however, properly infers that

the highly destructive properties of such substances as arsenic or corrosive sublimate, must have been well known to the prisoner, if an adult, by common repute.

It need hardly be observed, that the *quantity found in the stomach* or viscera, can convey no idea of the quantity actually administered; since more or less of the poison may have been removed by violent vomiting and purging as well as by absorption. But the quantity found in the stomach, even after a portion has been thus lost, is often more than sufficient to destroy the life of a human being. It is singular that, notwithstanding this very obvious cause for the removalof a poison from the stomach, barristers should so frequently address the inquiry to a medical witness—whether the quantity of poison found in the viscera was sufficient to cause death? Whether this question be answered in the affirmative or negative, is a matter which cannot at all affect the case, since either no traces of poison, or but a very small portion, may be found in the viscera, and yet the deceased may have assuredly died from its effects. Absorbed arsenic, as it exists in the tissues, is never found except in very minute proportion. (See ante, p. 30.) Thus, then, whether much or little be detected, the object of this question is not very apparent; since the fact of death having been caused by poison does not, in the least degree, rest upon the precise quantity which happens to remain in the dead body. It has been truly remarked by Orfila, in regard to arsenic, and it equally applies to all poisons, that that portion which is found in the stomach *is not that which has caused death;* but the *surplus* of the quantity which has produced fatal effects by its absorption into the system. The inquiry should therefore be directed to the probable quantity of poison *taken;* not to how much remains in the body.

This question is one of more importance than may at first sight appear. There is scarcely a trial for criminal poisoning, in which it is not put to a medical witness, either by the judge, or the counsel for the prosecution or defence. Supposing poison to be found in the stomach, but not in sufficient quantity to destroy life,—is it therefore to be assumed that the person did not die from its effects? This would be equal to laying down the doctrine, in face of the most indisputable evidence to the contrary,—that poisons, when taken into the body, are never liable to be expelled by vomiting or purging, or to be removed from the stomach by absorption! The real object of the toxicologist is to discover the poison by clear and undoubted evidence. If more than sufficient to cause death be discovered, then the dose must have been larger than was necessary; but if this proof be always required, what is to become of those cases of criminal poisoning in which the prisoner administers a dose only just sufficient to destroy life, or in which the deceased, by the strength of his constitution, happens to survive the effects for some days or weeks, and ultimately dies of exhaustion? No poison would be detected under these circumstances. The accused parties should either be acquitted, or one cannot see the object of putting such a question in any case. Orfila has most completely demonstrated the fallacy of this objection to medical evidence, and the danger of a Court of law relying upon it. (See Ann. d'Hyg. 1845, i. 347; also Toxicologie, ii. 731.)

Delicacy of analysis.—In conducting an analysis, the smallest possible quantity of the suspected liquid or solid should be used. If all were used at one operation, doubts might afterwards arise in the mind of the analyst, which it would be out of his power to remove. By care and ordinary precaution, a few grains will give results as satisfactory as those obtained from several ounces; and there is this additional advantage, that a portion is saved for the corroborative experiments of other analysts, or for correcting those which may have been previously performed. As a general rule, only one-half of the substance delivered for analysis should be examined. With respect to the minute quantities of poison which may be detected by chemical processes, some remarks will be made hereafter. It is, indeed, fortunate for the ends of justice, that the poisons which are commonly selected by criminals, may be discovered when existing in proportions so small as to excite wonder and incredulity in those who are not much acquainted with this department of science. The opinion of an experimentalist as to the *presence* of poison is never based upon the *quantity* actually found; for the results may be as infallible with a grain, or even the hundredth part of a grain of some substances, as with many ounces. All tests have a limit to their action; and when they act obscurely, or cease to act, the witness is bound to state that the chemical evidence has failed. Arsenic may be, however, safely inferred to be present when we obtain a quantity of the metal scarcely ponderable in the most delicate balance. We might go on with the experiment, and obtain from other portions still larger quantities of the metal; but the evidence of the presence of the poison would not be, chemically speaking, rendered more conclusive. A toxicologist merely obtains sufficient to enable him to speak safely to the actual presence of the substance: —what the weight or other physical properties of the quantity so obtained, may be, is a matter of no moment to him. It is customary for some medical witnesses to say that they only obtained *feeble evidence* of a poison by the application of tests. The use of these terms is liable to give rise to an erroneous impression. Either there is chemical proof of the presence of poison, or there is not:—the law knows of no intermediate stage of evidence, nor will it accept it as proof; and a witness will assuredly expose himself to a severe cross-examination who makes use of this ambiguous language upon a question of such vital importance. The tests may act upon a very *small* quantity—but the results should not in any case be doubtful; they must be certain and decided, or they are worth nothing. The quantity of poison to which the tests are applied, is left entirely to the judgment of the witness. It is necessary to observe in this place, that as the results of the action of tests depend in most cases upon the production of *coloured* precipitates, no reliance can be placed upon experiments which are performed by *artificial* light. Clear and open daylight is indispensably necessary to the analyst. By artificial light white precipitates acquire a yellowish tint; yellow precipitates appear white; red, brown; and in some instances the action of a test is completely obscured. Counsel would be fully justified in objecting to any analysis based upon results obtained under these circumstances, where the opinion of the analyst is derived from a change of colour.

Poisons in tests and apparatus.—If a practitioner has not been in the habit of analysing poisons, it is advisable, before he commences the analysis of the substance handed to him, that he should operate several times upon a portion of the same kind of poison, as that which is suspected to have been administered. In the employment of chemical tests, it is especially necessary to determine that they are pure before the analysis is commenced. Arsenic may be contained in the sulphuric or muriatic acid, or in the zinc, used in an analysis of a substance suspected to contain this poison; and sulphuric acid may be pronounced to be present in the stomach when it may have been contained in the nitric acid employed in the analytical process. Solutions of potash or soda, or their carbonates, if kept in flint-glass bottles, speedily acquire an impregnation of lead, and a suspicion might thus arise that a poisonous salt of this metal was present in a liquid to which they had been added as tests. Iron or metallic vessels (excepting platina or silver) should never be employed in the analysis of the viscera, whether in the humid or dry way. The iron of which these vessels are made almost always contains traces of lead, copper, tin, and arsenic. The use of Hessian crucibles is also objectionable in the incineration of the dry viscera for the fixed metals. The clay of which these crucibles are manufactured is very impure, and among other metals it yields especially traces of lead and copper. The vessels used in these experiments, should be made of white porcelain or platina. In all cases it is proper that the analyst should test his tests. A proceeding of this kind inspires confidence in the chemical evidence.

Danger of premature opinions.—During the examination of a suspected substance, a practitioner is often pressed to give an opinion respecting its nature before the steps of the process are complete. This may arise from the anxiety or curiosity of those who are interested in the proceedings. There is a rule, however, which it appears to me should always be followed on these occasions; namely, that no opinion whatever should be expressed until the *whole* of the analysis is complete. It often happens in the hands of the ablest analyst, that the last steps of a process, lead to a result very different from that which was anticipated at the commencement. The truth is, it is not by one character, but by many, that a poison is identified; and, therefore, a suspicion derived from a few incipient experiments, is very likely to be overthrown by continuing the investigation. In the *Boughton case*, Dr. Rattray gave an opinion in the first instance, that the poison administered to the deceased was arsenic; but he subsequently attributed death to laurel-water. A case occurred within my knowledge, where arsenic was pronounced to be present when sulphuric acid was really the poison. In another case tried at the Kingston Assizes in 1832, the medical witness admitted that, at the coroner's inquest, he stated the poison to be arsenic, but by subsequent experiments he found that it was oxalic acid. And in a case which has but recently occurred (February, 1845,) the poison was at first stated to be oxalic acid, but on a more careful examination, it was shown to be arsenic! Coroners are not sufficiently careful in selecting persons to conduct analyses of this kind: hence it is by no means surprising that such mistakes should be frequently made.

This mistake respecting the nature of a poison not merely impedes the course of justice, by throwing a doubt upon evidence which ought to be, beyond all question, clear and satisfactory; but it seriously affects the reputation of a witness. It entirely arises from his giving an opinion before he is justified by the facts in so doing. It is, I think, a well-marked line of duty to be pursued on these occasions;—1. That no opinion should be formed from a *few* experiments: and 2. That no opinion should be expressed until the analysis is *completed*. It is obvious that, if a man be compelled to admit in cross-examination at a trial for poisoning, that he has once been mistaken on a question so important, and requiring so decided an answer, a jury may be easily induced to believe that the witness may have made a second mistake, and that his then positive opinion is of no more value than that which he first expressed, and afterwards retracted. On the danger of trusting to an imperfect chemical analysis, see Annales d'Hygiène, 1829, ii. 405; xxvi. 399; xxix. 103, 474.

Fallacies in chemical analysis.—The improved character of medical evidence is perhaps in no instance more strongly manifested, than in the facility and certainty with which, in careful hands, the chemical analysis of poisons is now conducted. A hundred years ago, chemical tests were either unknown, or so improperly applied, that really innocent persons charged with the crime of poisoning, incurred great risk of their lives, in consequence of the chemical mistakes into which the medical witnesses of those days were so apt to fall. In some cases, although right in their inferences respecting death by poison, they were entirely wrong in their analyses. The evidence of Dr. Addington, a most eminent physician of his day, at the trial of *Miss Blandy* (Oxford Assizes, 1752,) for the murder of her father by arsenic, furnishes a striking instance of a series of chemical errors. Dr. Paris has very properly observed, that not a single substance employed by the witness could be regarded as a test for arsenic: he inferred the presence of the poison from certain chemical decompositions which could not possibly be ascribed to it. I quote the following extract from the report of the trial. Dr. Addington is asked:—"Why do you believe it to be white arsenic? For the following reasons—1st. This powder has a milky whiteness; so has white arsenic. 2d. This is gritty and almost insipid; so is white arsenic. 3d. Part of it swims on the surface of cold water like a pale sulphureous film, but the greatest part sinks to the bottom, and remains there undissolved; the same is true of white arsenic. 4th. This thrown on red hot iron does not flame, but rises entirely in thick white fumes, which have the stench of garlic, and cover cold iron just held over them with white flowers; white arsenic does the same. 5th. I boiled ten grains of this powder in four ounces of clean water, and then passing the decoction through a filter, divided it into five equal parts, which were put into as many glasses. Into one glass I poured a few drops of *sal ammoniac;* into another some of the *lixivium of tartar* (carbonate of potash;) into the third some *strong spirit of vitriol;* into the fourth some *spirit of salt;* and into the last some *syrup of violets.* The spirit of sal ammoniac threw down a few particles of a pale sediment; the lixivium of tartar gave a white cloud, which hung a little above the middle of the glass; the spirits of vitriol and salt made a considerable precipitation of a lightish-coloured substance, which, in the former, hardened into glittering crystals, sticking to the sides and bottom of the glass; syrup of violets produced a beautiful pale green tincture. Having washed the saucepan, funnel, and glasses used in the foregoing experiments very clean, and provided a fresh filter, I boiled ten grains of white arsenic, bought of Mr. Wilcock, druggist in Reading, in four ounces of clean water; and filtering and dividing it into five equal parts, produced with them just as I had done with the former decoction. There was an exact similitude between the experiments made on the two decoctions; they corresponded so nicely in each trial, that I declare I *never saw any two things in nature more alike*, than the decoction made with the powder found in Mr. Blandy's gruel, and that made with white arsenic. From these experiments, and others which I am ready to produce if desired, I believe that powder to be white arsenic." (Smith's Analysis of Medical Evidence, 202.) It would now be a difficult problem to determine what was the nature of the mixture to which the witness applied his tests. It is probable from the results, that it was a preparation of lead: there was certainly not the slightest evidence of the presence of arsenic, so far as these experiments went, either in the powder taken from the gruel, or in that actually sold for arsenic by the druggist! The female charged with the crime was convicted and executed; but fortunately her conviction did not depend upon this extraordinary specimen of forensic chemistry.

In the chemical evidence given upon some modern trials, there has been one source of error against which the witness should sedulously guard himself. It is not sufficient to apply tests to show negatively that a suspected poison is contained in a substance requiring analysis, but its nature must be clearly demonstrated by a series of affirmative results. A case occurred a few years since, in which oxalic acid was inferred to have been the substance which destroyed life, because the liquid was acid, and no other poison could be detected in it! This kind of evidence can never amount to proof. It is better to express no opinion at all, than to base our conclusion on probabilities.

One of the most serious difficulties to an analyst, is that which arises from the fallacies to which the application of the best tests is exposed. I do not now refer to the presence of impurities in the tests employed, but to the important fact that many substances, poisonous and not poisonous, yield the same or similar results with the same test. Hence if one test or one process be relied on as the basis of chemical evidence, the medical witness would fall into a dangerous mistake. We are perhaps hardly yet acquainted with all the fallacies to which *individual* tests are exposed:—the extension of chemical science is daily adding to their number by bringing out an analogy of properties where it could not have been suspected to exist. It is thus that selenic or fluosilicic acid may be mistaken for the sulphuric—the racemic or paratartaric for the oxalic acid—cadmium or antimony for arsenic—uranium for copper—bismuth for lead, with many other examples of the like nature. The usual means of avoiding a difficulty of this kind is to employ *several* tests or processes, and never to rely upon the action of *one*. It must be remembered that if the poison be really present, not a single test ought to fail in its reaction except from circumstances which it would be easy to understand and explain. Under each poison the objections hitherto known as applicable to the tests will be stated, and the means of avoiding fallacy from their use, explained.

When but small traces of poison are discovered, and large quantities of materials have been used for its extraction, as in what Dr. Christison properly designates the "enthusiastic" analyses of some modern French medical jurists, it would be unsafe to base any conclusion upon the results. Thus, in some recent French trials, the medical witnesses have not hesitated to boil up and evaporate the whole of the human body with many gallons of water and acids in large iron cauldrons, and have inferred that the individual died from arsenic because they had detected in his remains, infinitesimal traces of the poison! The dramatic effect of these gigantic researches, was probably never more strikingly displayed than in the well-known case of Laffarge. The body of the husband was undergoing evaporation in large iron vessels outside the Court, while the wife was on her trial for the murder within! The quantity of sulphuric acid, nitric acid and nitre, which must be used on such occasions, is so great, that there is good reason to suspect the probable introduction of small traces of poison ab extra. A jury would, undoubtedly, be fully justified in rejecting chemical evidence procured by such means: and in any similar case the witness ought to be called upon to state whether he has previously examined for poison, *equal quantities* of the substances which he employed in the analysis. Evidence of a much less ambiguous character has been frequently rejected by a criminal Court in England.

CHAPTER XI.

TESTS AND APPARATUS REQUIRED FOR THE ANALYSIS OF POISONS—LIST OF TESTS—PROCESSES FOR PREPARING CERTAIN TESTS—RULES FOR DETERMINING THE NATURE OF MINERAL POISONS. ANALYTICAL TABLES OF THE MINERAL AND VEGETABLE ACIDS. TABLE OF ALKALINE POISONS AND THEIR SALTS—RULES FOR DISCRIMINATING COLOURED AND COLOURLESS MINERAL POISONS—TABLES OF THE ACTION OF FERROCYANIDE OF POTASSIUM AND SULPHURETTED HYDROGEN GAS UPON METALLIC SOLUTIONS. ON THE VARIOUS PROCESSES FOR DETECTING POISON IN THE TISSUES OF THE BODY. RULES FOR DRAWING UP MEDICO-LEGAL REPORTS.

MANY of the tests employed in the analysis of poisons may be procured in a sufficiently pure state for immediate use. There are others, however, which it will be proper for the analyst to prepare for himself. I believe that the following list comprises all those preparations which are necessary for conducting the analysis of the poisons described in this work.

ACIDS. Sulphuric, Nitric, Muriatic, Oxalic, Tartaric, Acetic.

ALKALIES. Potash, Soda, Ammonia, and their Carbonates. Calcined Carbonate of Soda. Lime.

Alkaline and Metallic Salts.—Nitrate of Barytes. Chloride of Barium.—These may be made by digesting the pure carbonate in the respective acids, and evaporating to crystallization. Chloride of Lime. Sulphate of Lime. Nitrate of Silver. Sulphate of Iron. Ferrocyanide of Potassium. Phosphate of Soda. Sulphate of Copper. Iodide of Potassium. Acetate of Lead. Corrosive Sublimate. Peroxide of Manganese. Carbonate of Barytes.

Black Flux.—Prepared by mixing thoroughly two parts of bitartrate of potash with one part of nitrate of potash, and projecting the mixture by small portions into a red hot crucible, until complete deflagration has taken place. The grey mass obtained should be pulverised, and kept from air in a well-closed bottle. This substance is used for the reduction of the compounds of arsenic. The bitartrate itself calcined, or well-dried oxalate of lime, will answer the same purpose. (See Soda Flux.)

Chloride (Ter) of Gold.—Dissolve gold foil at a gentle heat, in a mixture of one part of nitric and two parts of muriatic acid. The solution may be afterwards diluted with its bulk of distilled water. Used to distinguish meconic from sulphocyanic acid.

Chloride (Bi) of Platina.—Dissolve slips of fine platina foil or platina filings, in a mixture of one part of nitric and two parts of muriatic acid, brought to a boiling temperature. Platina must be added, until no further action ensues. This is a useful test for potash.

Chloride of Tin.—Obtained by digesting pure tin in strong muriatic acid at a gentle heat, until no more is dissolved. A piece of metallic tin should always be kept in the solution. A useful test for Gold and Mercury.

Cyanide of Potassium.—This is a most useful reducing agent for the insoluble sulphate of barytes, arsenic, and other poisons. It is prepared by heating to strong redness dried ferrocyanide of potassium, reduced to a fine powder, in an earthen retort, with the beak loosely closed. The semifused mass in the retort, which must not be exposed to air until quite cold, is reduced to a fine powder, placed in a funnel, moistened with alcohol, and *cold* water poured over it (hot water leads to its reconversion to ferrocyanide.) The first concentrated colourless solution which passes through the funnel, is rapidly evaporated to dryness in a porcelain dish, and bottled. The white residue is a mixture of cyanate of potash and cyanide of potassium. It should be kept from air, in a dry place.

Hydrosulphuret of Ammonia.—Pass sulphuretted hydrogen gas by means of a bent tube from a long-necked flask into equal parts of a solution of pure ammonia and water, until the liquid is saturated with the gas. The solution must be preserved in a green-glass bottle. This is an important test for the detection of metallic poisons. When well made, it ought to give no precipitate with sulphate of magnesia.

Iodic Acid.—Digest Iodine in the strongest Nitric Acid (Sp. gr. 1.52) in a retort over a sand-bath, and repeatedly wash down with the acid, the iodine that may sublime. This process requires many hours for its completion. When there is no further action, pour off the liquid, and evaporate to dryness. Iodic acid is left as a colourless solid. This test serves to distinguish morphia from the other alkaloids, to detect sulphuric acid in articles of clothing, and to indicate the presence of sulphurous acid.

Oxalate of Ammonia.—Prepared by neutralizing a strong solution of Oxalic acid, with Sesquicarbonate of Ammonia, and evaporating at a low temperature to crystallization. Should the salt become acid by evaporation, add a little ammonia.

Sesquichloride (Permuriate) of Iron.—Dissolve red (per) oxide of iron in muriatic acid. It may be neutralized for the purpose of a test by the addition of a small quantity of potash. Used as a test for morphia and its salts.

Soda Flux.—Calcine, in a covered platina crucible, crystallized acetate of soda reduced to a fine powder. The charred mass may be afterwards pulverized. It does not deliquesce like the black flux, and is a good reducing agent. Neutralize tartaric acid by carbonate of soda,—evaporate to dryness, and incinerate in a covered platina crucible. The black residue (carbon and carbonate of soda) is an excellent reducing agent for arsenic.

Sulphate of Strontia.—This salt in solution, is sometimes used as a test for the salts of barytes. It may be made by digesting pure carbonate of strontia in diluted sulphuric acid. It is not very soluble in water, in consequence of which, when employed as a test, it must be used in comparatively large quantity.

Sulphuretted Hydrogen Gas.—This should always be employed in the state of gas, and not dissolved in water. It may be prepared by gently heating in a retort, or still better in a flask with a bent tube, sulphuret of iron with three parts of water and one part of strong sulphuric acid. The heat evolved on mixture, liberates the gas abundantly. It should be passed into distilled water for a short time before use. Care must be taken not to distil over the contents of the retort, if a retort be employed. This gas precipitates the salts of most metallic poisons, some completely, others partially. In an unknown case, the suspected solution into which it is passed should neither be acid nor alkaline, but neutral.

Sulphuret of Iron.—Heat a bar of iron to whiteness, and rub on its surface a stick of sulphur. Collect the sulphuret which falls in a state of fusion, in a vessel of cold water placed beneath. Dry it, and keep it closely bottled. This preparation serves for the purpose of making sulphuretted hydrogen gas.

Test Papers.—*Litmus* paper for acids.—This may be made by saturating unsized paper (free from lime) in a strong infusion of litmus (about one ounce to half a pint of boiling water) and drying it in a place entirely free from acid vapours. If not of a good blue colour, the paper may require to be dipped a second time. It should be kept from air and light. *Rose* paper for alkalies. This is made by saturating unsized paper two or three times in a strong infusion of red roses (about two ounces of petals to a pint of water) and drying the paper quickly. It should be kept from air and light, and in a dry situation.

Miscellaneous Articles.—Copper filings.—The finest copper gauze.—Thin copper-foil.—Copper-wire.—Tin-filings.—Tin-foil—Zinc-foil, very thin, —Gold-leaf. Gold-foil, such as is used by dentists: in this state it serves for the detection of mercurial poisons. Reduced silver. Platina-foil. Platina wire. Platina crucible and cover. Platina cup: these two vessels may each have a capacity of about two fluid-drachms. Small glass tube (about two pounds) varying from one-fourth to one-eighth of an inch in the bore. This tube, which serves for the making of small reduction-tubes, and numerous other purposes, should be very thin. Watch glasses. Test-tubes (thin)—Glass plates. Florence flasks. Large and small retort and receiver. Filtering paper. Spirit lamp. Charcoal powder. Animal charcoal. Alcohol. Ether. Litmus cake. Sulphate of indigo. Glazed card (lead.)

In pursuing an analysis, the following precautions ought to be observed: 1. All the apparatus should be perfectly clean; when metals are to be reduced, the glass tubes and fluxes should be warm and dry. 2. The solutions of the tests should be concentrated. This will give a known and definite strength which will regulate the quantity to be employed. 3. Before employing the tests, they should be tried for the ordinary impurities which they are liable to contain.

Rules for the Analysis of Mineral Poisons. One of the most difficult problems, which a medical jurist has to solve in relation to poisons, is that which is commonly left untouched in works on Toxicology; namely, what steps are to be pursued in order to determine the nature of a suspected poisonous substance. It is easy to verify, by the application of chemical tests, the nature of a mineral poison, when we know or really suspect what it is; but all who have exercised themselves in these matters must have felt the difficulty, amidst the multiplicity of tests, to make a selection, and apply them in particular cases. Assuredly, if experiments of this kind be conducted at random, the whole of the suspected substance is likely to be expended without any satisfactory results being obtained. Hence the necessity arises of adopting some rules of generalization, so that not only may the trouble attending an analysis be diminished, but a result more speedily obtained. If the poison has been taken and caused death, the analysis may, as it has been elsewhere stated, be assisted by ascertaining how soon after taking it, the symptoms occurred; their nature, the period at which the person died, and other circumstances of the like kind:—indeed, it has been already observed, that these very facts may disprove the suspicion of poisoning, and render a chemical analysis wholly unnecessary. But the substance may not have been taken, and in this case we can only proceed by chemical rules. In respect to the generalizations about to be made, I must observe that they only apply to the chemical analysis of those mineral substances which are most commonly employed as poisons; or otherwise it is obvious, that the whole department of mineral chemistry would require to be introduced. If any exception be made in this respect, it will be in relation to some very common substances, which closely resemble the mineral poisons in their physical or chemical properties. After all, the great difficulty in medical jurisprudence consists not so much in distinguishing poisons from other substances, as in distinguishing them from each other; since the cases in which a chemical analysis is chiefly demanded, are those where the substance taken, has given rise either to serious symptoms or to death. Some substances in the subjoined tables may not be regarded as poisons:—that is a point which does not require to be here discussed—(see ante, p. 15)—it is sufficient for a medical jurist to know that these reputed innocent substances have actually caused death; and therefore they may again come before him for a chemical examination.

I. Table of the acid poisons.

cids.	Nit. Barytes.	Nit. Silver.	Sulph. Lime.
Muriatic acid . . .	. . . . . .	precipitate insol. in nitric acid.	
Nitric acid			
Nitromuriatic acid .	. . . . . .	precipitate insol. in nitric acid.	
Nitrosulphuric acid	precipitate insol. in nitric acid.		
Oxalic acid. . . .	. . . . . .	precipitate sol. in nitric acid.	precipitate sol. in nitric acid.
Sulphate of Indigo. Sulphuric acid .	precipitate insol. in nitric acid.		

Nitric acid would be known by its action on copper:—nitromuriatic acid by its property of dissolving gold. For other and more special details, I must refer to the sections on individual poisons. Three tests only have been selected which, taken together, act differently, with the seven acid poisons in the table. Thus, if we obtain in a clear acid liquid, by the addition of nitrate of silver and sulphate of lime, white precipitates, soluble in nitric acid, there is every reason to believe that oxalic acid is present. The proofs required to establish this presumption will be found described under this poison.

The next table relates to the effects of various reagents on the four principal vegetable acids.

II.—TABLE OF VEGETABLE ACIDS.

Reagents.	Oxalic.	Tartaric.	Citric.	Acetic.
Heat	Volatile	melts and burns—red flame—carbon left	melts and burns—yellow flame, but little carbon left	liquid—no residue on evaporation.
Solution evaporated on plate glass	distinct four-sided prisms	irregular plumose crystallization	irregular stellated crystallization	no residue.
Lime water	precip. insol. in water and the acid	precip. sol. in water and the acid	slow precip. (if concent.) sol. in water and the acid	no effect.
Baryta water	precip. easily sol. in the acid	no effect	no effect	no effect.
Potash	precip. if acid in excess (Binoxalate)	precip. if acid in excess (Bitartrate)	no effect	no effect.
Sulphate of Lime	precipitate	no effect	no effect	no effect.
Chloride of Calcium	abundant precipitate	no effect	no effect	no effect.
Nitrate of Silver	abundant precipitate	precip. if concent.	no effect	no effect.
Sulphate of Copper	precip. if concent.	no effect	no effect	no effect.
Acetate of Lead	precipitate	precipitate	precipitate	no effect.

2. **Alkaline Poisons.**—In respect to the *alkaline* poisons and their salts, we may observe,—1. They are all white solids (some of them crystalline,) except the alkaline sulphurets. 2. The greater number are readily dissolved by water; the solutions having an acid, alkaline, or neutral reaction with test paper. 3. The solution or the solid diffused in water, undergoes no change when exposed to a current of sulphuretted hydrogen gas:—some of the acid salts throw down sulphur from the hydrosulphuret of ammonia, and alum gives an abundant precipitate of alumina with this test. 4. Carbonate of potash gives a white precipitate with baryta and its salts, while it does not affect potash, soda, and ammonia, or their salts. Lime and strontia are introduced

III.—Table of the Alkaline Poisons and their Salts.

Alkalies and Salts.	Water.	Re-action.	Chlor. Platina. Tartaric Acid.	Sulphuric Acid.	Oxalic Acid.	Sulph. Lime.
Alum	soluble	acid				
Ammonia	sol.	alkaline	precip.			
Sesquicarb.	sol.	alk.	precip.	efferves.	efferves.	precip.
Barium, Chloride	sol.	neutral	no precip.	precip.	. . . .	precip.
Baryta	sol.	alk.	no precip.	precip.	precip. sol.	precip.
Acetate	sol.	neutral	pre. T. A. sol.	Acet. Ad. evolv.	precip. sol.	precip.
Carbonate	insol.	. . . .	efferves.	efferv. ins.	efferv. partly sol.	
Nitrate	sol.	neutral	no precip.	precip.	. . . .	precip.
Potash	sol.	alk.	precip.			
Binoxalate	sol.	acid	. . . . . .	. . . .	. . . . . .	precip.
Bitartrate	sol.	acid				
Carb. and Bicarb.	sol.	alk.	precip.	efferves.	efferves.	precip.
Nitrate	sol.	neutral	precip.	Nc. Ad. ev.		
Potassium, Bromide	sol.	neutral	pre. T. Acid	Brom. ev.		
Iodide	sol.	neutral	pre. T. Acid	Iodine ev.		
Sulphuret	sol.	alk.	precip.	Sy. hyd. S. pr.	Sd. hyd. S. pre.	
Soda	sol.	alk.				
Carb. and Bicarb.	sol.	alk.	. . . . . .	efferves.	efferves.	precip.
Sodium, Chloride	sol.	neutral	. . . . . .	Mc. Ad. ev.		
Lime	sol.	alk.	pre. T. A. sol.	no precip.	precip.	no precip.
Salts of Lime	sol.	neutral	. . . . . .	pre. if concent.	precip.	no precip.
Strontia	sol.	alk.	. . . . . .	precip.	precip.	no precip.
Salts of Strontia	sol.	alk.	. . . . . .	precip.	precip.	precip.

N. B. The substances in italics are not commonly ranked among poisons.

not as poisons, but to show how they may be distinguished from baryta, for which they are very liable to be mistaken. 5. These salts are distinguished from the metallic irritants except tartar emetic (Tab. V.,) in not being precipitated by a solution of ferrocyanide of potassium.

METALLIC POISONS.—The metallic irritant poisons may be divided into two groups; those which are *coloured*, and those which are *colourless*. A slight attention to colour, will enable us to distinguish many of these bodies from each other, and by this criterion, if properly observed, we may determine at once the steps of the analysis to be pursued. In the first place, it will be noticed that the greater number of them are quite insoluble in water: those which are soluble have the letter *s* prefixed. Some are not at all changed in colour when treated with sulphuretted hydrogen or hydrosulphuret of ammonia,—these are marked with the letter *a;* the other coloured substances are variously affected, being generally turned brown or black. The substances printed in italics, are not commonly ranked as poisons:—they are introduced in order that the coloured poisons may not be mistaken for other coloured substances which resemble them. The four colours are yellow, blue, green, and red:—in any two substances of either colour, there is scarcely the same shade, so that when once compared, they may be in general easily distinguished.

IV.—METALLIC IRRITANTS.—COLOURED MINERAL POISONS.

Yellow.	Blue.	Green.	Red.
Masicot. (Oxide of lead.)	s Sulph. Copper.	Scheele's green. (Ars. Copper.)	Red Oxide Mercury
Turner's yellow. (Oxychlor. Lead.)	s Nitrate Copper.	Schweinfurth gr.	Nitric Oxide.—
	s Ammoniuret.—	(Ars. and Acet.	Cinnabar.
a Orpiment.	Verditer.	Copper.)	a Vermilion.
Turbith mineral.		Chloride. Copper.	
Chromate of Lead.		Brunswick green. (Oxychloride.)	Litharge.
s —— of Potash.			Minium.
s Alkaline Persrts.		s Acetate Copper.	a Realgar.
		s Subacetate —	s Bichromate Pot.
		Carbonate —	s Alkaline Sulphurets.
Ammoniuret Iron.		s Sulph. Iron.	
Iodide of Mercury	*Smalt.* (*Ox. Cobalt*)		
a *Persulphrt. of Tin*	*Prussian Blue.*		*Biniodide Mercury*
a *Sulphrt. Cadmium*	*Indigo.*	*Oxide of Chrome.*	*Peroxide of Iron.*
s *Ferrocyanide Pot.*		s *Salts of Nickel*	a *Oxysulphuret of Antimony.*
Perox. Uranium.			

COLOURLESS METALLIC POISONS.—The colourless metallic irritant poisons are very numerous, and it is commonly among these, that the greatest difficulty is experienced in an analysis. They may, however, be distinguished from each other by treating them with certain re-agents (see Table V.:)—but these are merely to be regarded as trial tests; *i. e.* to point to the particular nature of the poison for the detection of which special tests are subsequently to be applied. The re-agents may be added either to the solids, or, if the mineral poison be soluble, to the solutions in water. A very convenient mode of employing the tests is to place a small quantity of each in a small white saucer,

V. METALLIC IRRITANTS.—COLOURLESS IRRITANT POISONS.

	Heat.	Water.	Sulphuretted Hydrogen.	Hydrosulphuret of Ammonia.	Potash.	Iodide of Potassium
Arsenic acid	fixed	very soluble	pale yellow pr.	yellow by evap.	soluble	
Arsenious Acid	volatile	sol. with difficulty	yellow prec.	yellow by evap.	soluble	
Bismuth, Subnitrate	yellow (fixed)	insoluble	black	black	. . . .	brown.
Calomel	volatile	insoluble	black	black	black	green yellow.
Corrosive Sublimate	volatile	soluble	black	black	orange yellow prec.	scarlet precip.
Emetic Tartar	carbonized	soluble	orange red	orange red	white p. sol.	
Lead, Acetate	carbonized	soluble	black	black	white	yellow.
Carbonate	orange	insoluble	black	black	white	white (on adding acetic acid yellow.)
Chloride	melts (fixed)	soluble	black	black	white	yellow.
Nitrate	yellow decompd.	soluble	black	black	white	yellow.
Subacetate	carbonized	soluble	black	black	white	yellow.
Sulphate	fixed	insoluble	black	black	white	yellow.
Mercury, Bicyanide	vol. decompd.	soluble	black	black	unaffected	white sol.
Nitrate	vol. decompd.	soluble	black	black	black	yellow.
Pernitrate	vol. decompd.	soluble	black	black	orange yellow prec.	scarlet.
White precipitate	volatile	insoluble	black	black	yellow by heat	orange.
Silver, Nitrate	fixed (melts)	soluble	black	black	brown	pale yellow.
Tin, Chloride	fixed	soluble	black	black	white p. sol.	
Dyer's Spirit	fixed	soluble	yellow	yellow	white p. sol.	
Zinc, Acetate	carbonized	soluble	white	white	white p. sol.	
Carbonate	yellow	insoluble				
Oxide	yellow	insoluble				
Sulphate	fixed	soluble	white	white	white p. sol.	

and then drop a few grains of the suspected solid in fine powder into the liquid. Any change of colour is immediately perceptible. These poisons are said to be colourless; but some of them have a slight tint, bordering on yellow.

It is impossible, in giving a summary of this kind, to represent the slight differences in respect to solubility and the colours produced by the various reagents; but these it is not difficult for one, moderately acquainted with chemistry, to appreciate, and the facts are all fully explained in the description of the poisons individually.

There is a very useful test, however, which enables us to distinguish the salts of many metallic poisons. This is the Ferrocyanide of potassium. I here subjoin a table, derived from experiments in which the different effects of this reagent on numerous metallic solutions are indicated.—(See next page, Table VI.)

There are four states in which a poison may be presented for analysis:—1, *solid* and unmixed with other substances; 2, pure and in *solution* in water. It is anticipated that the preceding tables will enable a medical witness to determine, without much difficulty, in these cases, the probable nature of the substance he is examining, and direct his analysis accordingly. It may not happen to be a poison: then the discovery of its nature must depend on his general knowledge of chemistry.

In order to illustrate the mode of applying these tables, let us suppose that we have a white powder which we find to be entirely volatile without residue by heating it on platina foil—that it readily dissolves in water, especially on boiling—that it is blackened when placed in contact with hydrosulphuret of ammonia—becomes yellow with caustic potash, and a bright scarlet with iodide of potassium. These results indicate CORROSIVE SUBLIMATE; and on turning to the description of that poison, other modes of testing will there be given, which will enable the operator to demonstrate conclusively the presence of chlorine and mercury. Again, we may have a white powder, also volatile without residue, scarcely soluble in water even on boiling, undergoing no change of colour in contact with hydrosulphuret of ammonia, until the ammonia has been lost by evaporation, or driven off by heat (when a rich organge-yellow residue remains,)—unchanged in colour by caustic potash, but easily dissolved by it on boiling, and unchanged in contact with iodide of potassium. These results point at once to ARSENIOUS ACID.

There are two other states in which the analysis of a poisonous substance may be required: 3, *solid* and mixed with organic solids, as arsenic in powdered coffee; and, 4, dissolved or suspended in *liquids* containing organic matter, as corrosive sublimate in wine, porter, beer, or milk. The last is a condition frequently requiring investigation:—the third stage merges in the fourth; since we are in general compelled to boil the poisoned solid in water, in order to ascertain its probable nature. Alcohol or ether may be occasionally substituted for water; in one case these liquids may serve to remove the poison from the organic matter, and in another, to dissolve the organic matter and leave the poison. Let us, however, assume that the poison is dissolved in an organic liquid: all its physical properties are lost, and we can now trust to chemical reagents alone. With regard to the soluble salts of copper, they never exist in an organic liquid in a far less than poisonous proportion, without giving to it a decidedly blueish or greenish colour, by which character, therefore a liquid containing a soluble cupreous poison may be commonly known. In order to distinguish the principal soluble metallic poisons, we employ sulphuretted hydrogen gas: if we except arsenic, all the metallic poisonous solutions are precipitated by hydrosulphuret of ammonia. The colour of the precipitate is different for different metals; and it is in this way that we may derive an inference of the kind of metallic poison present.

TABLE VI.—METALLIC IRRITANT POISONS. ACTION OF FERROCYANIDE OF POTASSIUM UPON THEIR SOLUTIONS.

Metallic Poisons.	Colour of Precip.	Observations.
ANTIMONY, Chlor. pure..	cream white	
impure	deep blue	from presence of iron.
Tartarized . . .	no precip.	
ARSENIC, Arsenious Acid	no precip.	
Arsenic Acid . .	no precip.	
Arsenite Pot. . .	no precip.	
Arseniate Pot . .	no precip.	
BISMUTH, Nitrate. . .	yellow-white	slowly green, by decomp. of ferrocyanic acid.
CADMIUM, Nitrate . .	white	gelatinous, like zinc.
COBALT, Chloride . .	dingy olive green	modified by red colour of solution.
COPPER, Sulphate . .	deep red	gelatinous.
GOLD, Terchlor. . . .	no precip.	liquid green, from decomp. of ferrocy. acid.
IRON, Persalts . . .	deep blue	
Protosalts . . .	white	becoming deep blue by exposure.
Green Sulph. . .	light greenish-blue	
LEAD, Acetate. . . .	cream white	
MANGANESE, Neut. Chlor.	white	if iron present, blueish-white.
MERCURY, Protonit. . .	white	becoming greenish from decomp. of ferrocyanic acid.
Pernit.	white	
Corr. Sub. . . .	white	
NICKEL, Nitrate . . .	pea green	resembling Scheele's gr.
PLATINA, Bichl. pure .	light fawn colour	greenish colour by decomp. of acid, rapid if concent. blue if iron is present.
Amm. Chlor. . .	no effect	
SILVER, Nitrate . . .	white	
TIN, Chloride. . . .	milk white	gelat. (brown by H. S. A.)
Perchlor.	white	(yellow by H. S. A.)
Dyers' Spirit . .		
URANIUM, Pernit . . .	rich red colour	resembling copper, but not gelatinous.
ZINC, Sulphate. . . .	white	gelatinous, blueish if iron is present.

Of the *yellow* and *orange-red* sulphurets it may be observed, that the sulphuret of arsenic is not dissolved by muriatic acid, even on boiling—those of cadmium and antimony are, however, easily decomposed by this acid—sulphuretted hydrogen is evolved, and soluble chlorides result. The white sulphuret of zinc is similarly affected. It is important for the analyst to remember, as one method of separating these metallic poisons, that sulphuretted hydrogen gas does not precipitate *acid* solutions of the *oxide of zinc*, *oxide of nickel*, *protoxide of cobalt*, *protoxide* and *peroxide of iron*, and *protoxide of manganese*.

A convenient way of testing an organic liquid, is to dip into it a slip of bibulous paper, and then expose this to a current of the gas produced in the tube; we thus get rid of the effect of colouring matter, but this method will only answer where the quantity of poison dissolved is moderately large. When there is no change of colour in the slip of paper under these circumstances, we may filter off one-fourth of the liquid, neutralize it by potash or acetic acid, according to whether it be acid or alkaline, and then pass into it, from a retort or bottle, a washed current of the gas. If the change of colour be not apparent in the liquid, it will be often perceptible in the froth or scum, which will readily assume a colour according to the nature of the metallic substance present. If the paper or froth should be turned of a *yellow* colour,—it may be owing to the presence of arsenic, cadmium, or tin. For obvious reasons, the presumption is generally in favour of arsenic,—a fact which will be apparent by the golden-yellow colour of the paper being immediately discharged on dipping it into a strong solution of ammonia. Should the yellow colour be owing to the presence of tin or cadmium, the organic liquid will be equally precipitated, or the paper will be stained by hydrosulphuret of ammonia. The yellow precipitate of arsenic is dissolved by ammonia, and not by strong muriatic acid; that of cadmium is dissolved by muriatic acid, and not by ammonia; that of tin is not readily dissolved by either reagent: but arsenic would be better known by boiling a portion of the liquid with muriatic acid and metallic copper.

VII.—METALLIC IRRITANT POISONS. ACTION OF A CURRENT OF SULPHURETTED HYDROGEN GAS, OR OF HYDROSULPHURET OF AMMONIA, UPON THEIR SOLUTIONS.

Yellow.	Orange Red.	Black or Brown.	White.	Green.
Arsenic (S.H.) Dyer's Spirit. (Persalts of Tin.) *Salts of Cadmium.* *Persalts of Uranium.* (yellow-brown.)	Salts of Antimony. Tartar Emetic.	Lead. Copper. Mercury. Bismuth. Protosalts of Tin. Salts of Iron. (Copperas) (H.S.A.) Nit. Silver. Chlor. Gold. *Nickel (H.S.A.)* *Cobalt (H.S.A.)* *Tellurium.*	Salts of Zinc.	Bichromate of Potash. Chromate of Potash.

If the slip of paper acquire a *black* or *brown* colour,—although there are many metals thus affected, the presumption is always in favour of lead or mercury: in the former case, the colour is instantaneously produced by the gas, in the latter (corrosive sublimate,) slowly. The salts of copper would be indicated by the green tint of the liquid, and by a metallic deposit on polished iron. It is very unlikely that any of the other metals should be present;—and with respect to the soluble salts of mercury and lead,—the last are known from the first by the organic liquid being abundantly precipitated by diluted sulphuric acid. The galvanic test either of gold and zinc or of zinc and platina, may be also applied. When zinc is present, no effect is produced on the slip of paper; and it is then necessary to pass a current of gas into a portion of the liquid,—the milky whiteness of the froth will indicate zinc. Antimony is peculiar in producing an orange-red colour, which is removable by caustic potash and strong muriatic acid, but not readily by ammonia. When by these experiments we have determined the probable nature of the poison, we may pursue the process recommended for the particular metal in a state of mixture with organic substances.

Should sulphuretted hydrogen gas produce in a neutralized and concentrated liquid no perceptible change, the poison, if any, may belong to those mentioned in Tables I. and II. If none of these be present, and there is no effect when the liquid is much concentrated by evaporation, there is probably not sufficient of the metallic poison present to allow of its separation and identification. There are some exceptions to this remark; as in the case of arsenic and antimony, which may be still detected by Marsh's or Reinsh's process,—of mercury, by gold and zinc; of copper, by iron, when sulphuretted hydrogen gas produces either no change at all, or the change is of an ambiguous character.

Mineral poison in the tissues.—The preliminary experiments hitherto made, presuppose that the poison exists in the viscera or their contents in a form which renders it easily soluble in water. Let us suppose, however, that no trace of poison can be detected by these eliminating tests in the suspected organic liquid, in the contents of the stomach, or in the tissues of the stomach, although they may have been boiled in water. Let us imagine that the stomach itself—the liver or spleen, has been produced for analysis, and no poison soluble in water can be extracted. The analyst is then compelled to resort to an entirely different method. The principle here consists in destroying all the organic matter, and rendering the tissues of the organ, with any poison that may be locked up in them, perfectly soluble. To accomplish this it is necessary to employ mineral acids. Orfila has recommended the drying of the animal substance, and its subsequent incineration with nitre; but the poison, if in small quantity, is likely to be lost by this operation. Nitric acid, also proposed by Orfila, is inconvenient on account of the large quantity of gaseous matter evolved during decomposition.

Sulphuric Acid.—M. Flandin first proposed carbonization by pure sulphuric acid. The organic substances, if liquid, are brought to dryness by evaporation, and then treated with one-third the weight of concentrated sulphuric acid. If the evaporating dish be weighed, and after the evaporation, it be again weighed with the residue, the quantity of sulphuric acid to be added, will be equal to one-third of the difference in weight. If the substance be solid, it is at once cut up into small pieces, and one-third of the acid added. A gentle heat should be at first employed, to prevent loss by projection; but when the organic matter is thoroughly dissolved in the acid, the process may be accelerated, taking care to keep the viscid mass well stirred. The dish should be equally heated at the sides. If the mineral poison is of a fixed nature, a full red heat may be employed: if volatile, like arsenic or mercury, it is necessary to stop short of a heat of redness, or there would be a serious loss: When the carbonized residue is thoroughly dried, it should be reduced to a very fine powder, and moistened with a mixture of three parts of nitric and one of muriatic acid: it is again evaporated to dryness, the residue treated with warm water three times successively, filtered, and concentrated by evaporation. With the blood and brain, the quantity of sulphuric acid required for perfect carbonization is equal to half the weight, while one-fourth will suffice for the lungs or the intestines. A preliminary trial on about an ounce of the dried organized matter will show what proportion of acid is really required. The liquid obtained should be clear and colourless; it may then be tested for the various poisons. It is better to carbonize the organic matter in several different processes, than to employ a large quantity at once. About eight ounces of the liver, or any of the dried organic tissues, will yield enough of the poison, if it be present, for evidence. The acids employed, should be tested for poison in the quantities in which they are used.

Muriatic acid.—The tissues may be rendered soluble, and the poison extracted in a form fitted for testing, by boiling the animal substance for two or three hours in a mixture of one part of muriatic acid and eight parts of water. This, which is commonly called the process of Reinsch, is well adapted for the separation of arsenic and mercury from thè soft organs, and there is less risk of the loss of poison. As some arsenic is lost by evaporation during the boiling with muriatic acid, Fresenius and Babo have advised that chlorate of potash, should be added to the liquid in order to prevent this loss. It is highly objectionable, however, to introduce many different substances into a liquid undergoing analysis for medico-legal purposes. The employment of a capacious retort and receiver, or of an alembic, will enable the operator to collect any arsenic that may escape with the muriatic acid. (See post, ARSENIC.)

In all the processes yet suggested for extracting poison from the tissues, there is likely to be a loss of arsenic when this poison is under investigation. MM. Flandin and Danger estimate this loss in the incineration by nitre at 8-10ths, in carbonization by nitric acid at 8-10ths, in the carbonization by sulphuric acid with nitrate of potash or soda at 4-10ths, in carbonization by sulphuric acid at 1-10th. (Galtier, Toxicologie, 1, 355.) M. Flandin thus takes a most favourable view of his own process; but it has been suggested that a serious loss of arsenic may occur in the decomposition of alkaline chlorides in the tissues, by sulphuric acid. (Annuaire de Chimie, 1846, 819.) My friend Dr. Geoghegan informs me that he has found carbonization by sulphuric, preferable to boiling with muriatic acid. Whichever plan may be adopted, we must remember that no tests are applicable until the whole of the animal matter is disintegrated, and the poison contained in it has been brought to a perfectly soluble condition; and as the quantity of poison in the tissues is always small, the most delicate tests must be employed, and the effect of the acids, used in the process upon the results must be borne in mind. It is also important, before employing these acids, that the analyst should be well assured of their purity, since arsenic is not unfrequently contained both in sulphuric and muriatic acid. These processes, it may be observed, are chiefly adapted to the detection of fixed mineral poisons in the tissues, and some modifications are required according to the nature of the poison. These, together with the tests, best fitted for employment under the circumstances, will be more fully described in treating of each poison individually.

Attempts have been made to devise a process which may be applicable to the detection of any metallic poison locked up in the tissues. One of the latest suggestions of this kind is that of Dr. Fresenius of Giessen. He considers that a method laying claim to general applicability should fulfil the following conditions:—"1. It must admit of detecting arsenic in every form in which this mineral can possibly exist. 2. It must not merely lead to the detection of arsenic, but also to that of the other metallic poisons. 3. It ought to preclude the possibility of confounding arsenic with other substances. 4. It must admit of detecting even very minute quantities of arsenic. 5. The method sought must enable us to obtain at least an approximate quantitative determination of the arsenic detected. 6. It must fulfil all these conditions by the most simple means." However desirable it may be to possess such a method as is here sketched out, the absolute necessity for it is not apparent. Arsenic may be most satisfactorily detected by processes which are not fitted for the detection of other metallic poisons. If each poison has its own particular process, and this is satisfactory so long as it is confined to its proper object, it is impossible to allow that the admissibility of chemical evidence in cases of arsenical poisoning, should be made to rest on the universal application of the same process, and with a like degree of certainty, to other poisons.

Out of the many processes suggested for the detection of arsenic in mixtures containing organic matter, there are, according Dr. Fresenius, only *four* which require to be mentioned. Arsenic may be separated,—1, as arseniate of lime; 2, as sulphuret; 3, from arsenuretted hydrogen; 4, by metallic copper. Of these methods, the second alone is recommended as fulfilling the conditions required, although it is obvious that the poison may be administered in the state of sulphuret, (yellow arsenic;) and therefore this, without some preliminary preparation, cannot fulfil the first condition required, *i. e.* of detecting arsenic in *every form* in which it can possibly exist. On the separation by lime nothing need be said, as it is now abandoned by toxicologists. Dr. Fresenius considers that the separation by arsenuretted hydrogen (Marsh's test) is absolutely inapplicable to the intended purpose, "because it does not admit of the separation of arsenic in every form in which this substance may exist." Further, it does not contribute to the detection of other metallic poisons; it contaminates the substance under investigation with zinc, which might itself have acted as the poisonous agent; it leads more easily than any other method to mistakes; and does not admit of any correct quantitative determination of the arsenic found. Marsh's test, it is allowed, permits the detection of minute quantities of arsenic in many cases in a very simple manner. Reinsch's process *i. e.* the separation by copper, is just as little adapted to the purpose, although, like the preceding test, it may serve to detect very minute quantities of arsenic. "The defects of this method are, it is alleged, that it does not admit of the detection of arsenic in every form in which the metal may/exist; that it does not lead to the detection of other metallic poisons; and that moreover the substance under investigation becomes contaminated by copper. Its success is impeded or prevented by the presence of many substances, such as nitrates, mercurial and other metallic compounds; so that the advantage of detecting even minute quantities of the poison can only be conditionally conceded to it. Finally, it does not allow of the quantitative determination of the arsenic present."

It appears to me impossible to assent to the validity of these objections, or to admit that we should be at all justified in entirely discarding the ingenious processes of Marsh and Reinsch, upon such grounds as are here adduced. These tests are fully equal to the separation of arsenic in all the forms in which *it is most commonly found in practice;* that they do not detect all other metallic poisons or that their operation on arsenic is occasionally rendered obscure by the presence of other substances, are objections which amount to nothing in the hands of those who limit the application of these tests to the purposes for which they were originally designed; and it is doubtful whether they are more liable to lead to fallacy in skilful hands, than the method by conversion to sulphuret. Neither Marsh's nor Reinsch's process will answer all the artificial conditions laid down as necessary for a universal method by Dr. Fresenius; but it is questionable how far it is just to measure the practical utility of these tests by such a standard as that which is here assumed.

Before converting the poison to the state of sulphuret, Dr. Fresenius considers the different plans which have been recommended for obtaining a clear solution, without loss of arsenic, from the organic mixture containing the poison. He gives the preference to the action of chlorine, as a decolourizing agent, and to the process of charring by sulphuric acid.

Fresenius's process.—The substances intended for examination, are, if formed of solid and coherent lumps, reduced into small pieces, and under all circumstances carefully intermixed. They are then put into a porcelain basin, and drenched with an amount of pure concentrated hydrochloric acid, either equal or superior to the weight of the dry substances, and with as much water as will give the consistence of thin pap to the whole mass. The basin is then heated in the water-bath, and chlorate of potash, in portions of about half a drachm, is added to the mixture at intervals of about five minutes, and until the contents of the basin have assumed a bright yellow colour, perfectly homogeneous, and a thin liquid consistency. When this point is attained, about two drachms more of chlorate are added, and the basin removed from the water-bath. When cool, the contents are placed on a filter of linen or paper, and are allowed to run off: the residue is washed with hot water until the liquid which passes (which is also collected) is no longer acid. The whole is then concentrated in the water-bath, during which process it changes from a bright yellow to a brownish tint, and a saturated solution of sulphurous acid is added to this residue until the smell of sulphurous acid is clearly perceptible. The whole mixture is then heated for about an hour, until the excess of sulphurous acid is completely expelled. A current of washed sulphuretted hydrogen is now slowly transmitted through this liquid for about twelve hours. The glass containing the precipitated sulphuret is lightly covered, and kept at a temperature of about 86° until the smell of sulphuretted hydrogen has disappeared. The precipitate which, besides sulphuret of arsenic, may contain organic matter and other metallic sulphurets, is collected and washed on a filter, and afterwards dried with it in a porcelain basin heated by a water-bath. Fuming nitric acid is then added drop by drop, until the whole is moistened; it is then evaporated to dryness. Pure hydrated sulphuric acid previously heated is then added to the residue, so as to moisten it uniformly: the mass is again heated in a water-bath for the space of from two to three hours, and finally in a sand-bath, at a temperature of about 300°, until the charred mass begins to crumble. The residue is digested in water, filtered, mixed with hydrochloric acid, and the filtered liquid precipitated by sulphuretted hydrogen. The precipitate thus obtained is drenched with a solution of ammonia, and the ammoniacal fluid is evaporated in a balanced porcelain basin, dried and weighed. The arsenic thus procured as sulphuret, may be quantitatively determined in the usual way. The residue, insoluble in ammonia, may be tested for lead, bismuth, copper, and mercury. The metallic arsenic is obtained from the sulphuret by heating it in a current of well-dried carbonic acid, with a mixture of carbonate of soda and cyanide of potassium as the reducing agent.

With the exception of the plan for removing the colour of the organic liquid containing arsenic, there does not appear to be any originality about this process. It is undoubtedly much more complex than some of the others for which it is recommended as a substitute; and unless it were conducted by one well versed in chemical manipulation, it might equally lead to error. The value of sulphuretted hydrogen gas as a precipitant has been long known, and is fully appreciated by toxicologists; but this does not preclude the employment of other, more ready and equally certain means for detecting arsenic. The process appears to be rather adapted for separating arsenic from its ores, than for detecting it as a poison in medico-legal cases. In practice we do not find the compounds of lead, bismuth, copper, and mercury, mixed with arsenic; and therefore it is useless always to have recourse to a process which invariably presupposes the admixture of these metals with the poison criminally administered. Even in cases of suspected compound poisoning, rare as they are, there is no difficulty in discovering the foreign metal mixed with arsenic by appropriate tests; and a Court of law requires to know whether arsenic was present and was the cause of death, rather than whether it was mixed with traces of bismuth or lead, a fact which, however interesting in a chemical, is wholly unimportant in a medico-legal view. It is doubtful whether by this process the absorbed arsenic could be detected in the soft parts of the body, with the same readiness as by those of Marsh or Reinsch.

MEDICO-LEGAL REPORTS.—One of the duties of the medical jurist is to draw up a report of the results of his examination: 1, in regard to symptoms; 2, in regard to the post-mortem appearances; and, 3, in regard to the results of an analysis. With respect to the two first divisions of the report, I must refer the reader to the chapter on the rules for investigating cases of poisoning (ante, p. 89.) I need hardly observe, that the time at which the person was first seen, and the circumstances under which the attendance of the practitioner was required, as well as the period of death, should be particularly stated. The hour, the day of the week, and the day of the month, should be invariably mentioned. Some medical witnesses merely state the day of the week, without that of the month, or vice versâ. At a trial this creates great confusion, by rendering a reference to almanacs necessary. The words yesterday, next day, &c. should never be used. The facts which it will be necessary to enter in the report, are specially stated under the heads of investigation (see p. 89.) If these facts be not observed in the order there set down, their value as evidence of the cause of death, or of the criminality of particular parties, will be entirely lost. In drawing up a report of symptoms and post-mortem appearances, the facts should be in the first instance plainly stated *seriatim*, without circumlocution, and in language easily intelligible to non-professional persons. A reporter is not called upon to display his erudition, but to make himself understood. If technical terms are employed, their meaning should be stated in parentheses. When a subject is thoroughly understood there can be no difficulty in rendering it in simple language; and when it is not well understood, the practitioner is not in a position to make a report. Magistrates, coroners, and barristers are very acute, and easily detect ignorance, even when it appears under the mask of erudition.

In recording facts, a reporter should not encumber his statements with opinions and inferences. His conclusions should be reserved until the end of the report. The language in which conclusions are expressed should be precise and clear. It must be remembered that these are to form a concise summary of the whole report, upon which the judgment of a magistrate or the decision of a coroner's jury will be ultimately based. They should be most strictly kept to the matters which are the subject of inquiry. Thus, they commonly refer to the following questions. What was the cause of death? What are the medical circumstances which lead you to suppose that death was caused by poison? What are the circumstances which lead you to suppose that death was *not* caused by natural disease? Answers to one, or all of these questions comprise in general, all that the reporter is required to introduce into the conclusions of his report.

The reporter must remember, that his conclusions are to be based only upon *medical* facts,—not upon moral circumstances, unless he be specially required to express an opinion with regard to them, when they are of a medico-moral nature. Further, they must be based only on what *he has himself seen or observed.* Any information derived from others should not be made the basis of an opinion in a medico-legal report. It is scarcely necessary to remark, that a conclusion based upon mere *probabilities* is of no value as evidence.

In drawing up a report on the *results of a chemical analysis*, the following rules may be borne in mind. A liquid or solid is reserved for analysis. 1. When and of whom or how received? 2. In what state was it received—secured in any way, or exposed? 3. If more than one substance received, each to be separately and distinctly labelled; appearance of the vessel, its capacity, and the quantity of liquid (by measure) or solid (by weight) contained therein. 4. Where and when did you proceed to make the analysis, and where was the substance kept during the intermediate period? 5. Did any one assist you, or did you make the analysis yourself? 6. Physical characters of the substance. 7. Processes and tests employed for determining whether it contained poison. All the steps of these processes need not be described; a general outline of the analysis will suffice. The magistrate may thus satisfy himself by an appeal to others (if necessary) to say whether the analysis has or has not been properly made. 8. Supposing the substance to contain poison,—is this in a pure state, or mixed with any other body? 9. The strength of the poison—if an acid or if it be in solution; in *all* cases the *quantity* of poison present. 10. Supposing no poison to be contained in it, what was the nature of the substance? Did it contain any thing likely to injure health or destroy life? 11. Could the supposed poisonous substance exist naturally or be produced within the body? 12. What quantity of the poison discovered, would suffice to destroy life, and how far is the dose likely to be modified by age or disease?

There are few reports in which answers to most of these questions, although not formally put, will not be required: and unless the whole of them be borne in mind by the operator at the time an analysis is undertaken, those which are omitted can never receive an answer, however important to the ends of justice that answer may ultimately become.

The results of analysis in the shape of sublimates or precipitates should be preserved as evidence distinctly labelled to correspond with the report, in small glass tubes hermetically sealed. In this way they may be produced for examination at the inquest or trial.

CHAPTER XII.

ON THE EVIDENCE OF POISONING FROM EXPERIMENTS ON ANIMALS—UNCERTAINTY OF RESULTS—CIRCUMSTANCES UNDER WHICH THIS EVIDENCE IS ADMISSIBLE—RECEIVED ON VARIOUS TRIALS FOR MURDER BY POISON—INJECTION-EXPERIMENTS. IS THE FLESH OF POISONED ANIMALS POISONOUS? ANIMAL FOOD POISONOUS IN CERTAIN CASES—RENDERED POISONOUS BY CERTAIN VEGETABLES.

SOME toxicologists have enumerated experiments upon animals as one among the sources of proof in cases of poisoning. This kind of evidence rests upon the assumption, that poisons act on man and the lower animals in the same way. The observations of Orfila, however, tend to show that this is partially true with only two domestic animals, namely, the dog and the cat:—in all other cases, the results by no means accord. With respect to experiments performed on dogs and cats, I quite agree with the opinion expressed by M. Devergie (Médecine Légale, ii. 457,) that they are in no case fitted to show the *doses* in which particular poisons are injurious or fatal to man—nor can they be safely trusted to prove the rapidity of action of different poisons. All that they are fitted for is to enable us to ascertain whether a particular substance be injurious to animal life or not, but nothing further. In *Donellan's* case, this kind of evidence was admitted to show the poisonous effects of laurel-water; and in *Freeman's* case, tried at Leicester in April 1829, experiments on animals were received as evidence to prove how speedily prussic acid, in certain doses, will destroy life. These experiments rather led to the presumption that the prisoner was guilty of the murder of a female by administering to her prussic acid, whereas, it was proved by circumstances, that he was innocent. An exclusive reliance upon results so obtained, is always liable to lead to erroneous medical evidence. In some experiments made on dogs by Dr. Ried and Dr. Simpson, they gave *an ounce* of Scheele's prussic acid to one animal, and it died in about a minute afterwards. Other dogs of the same size, to which about *six drops* of the same acid, from the same bottle, were given, died in the same period of time; although the dose in the last case was only one eightieth part of the quantity given in the first experiment. The contractility of the heart was in none of the cases much impaired. (Ed. Med. and Surg. Journal, Oct. 1836, p. 500.) From these and similar experiments, it is evident that no fair inference can be drawn of the relative effects of prussic acid on man and animals; for there is no agreement as to the action of the poison on the latter. Doses so widely differing from each other were found to kill dogs of similar size and strength within the same period of time.

When the question is merely, whether a suspected substance administered to another is or is not poisonous, then we may occasionally be justified in resorting to this kind of evidence, in order to determine the fact. Most of the common poisons are, however, capable of having their presence easily demonstrated by a chemical analysis; and the properties of the substance will be thereby known. But evidence of this description may be sometimes accidentally obtained, and then it will often dispense with a chemical analysis of the vehicle of the poison; indeed, it may supply proof when no poison is discovered in the body of the deceased. An intelligent barrister related to me the following case which he was engaged in prosecuting on the Western Circuit some years since. A woman poisoned her husband with arsenic mixed in soup: and after the deceased had made a full meal, she threw the remainder out of a window into a farm yard, thereby thinking to defeat all attempts at discovering the means which she had adopted to destroy her husband. It happened at the time, that a pig and several fowls were feeding under the window, and they ate up what fell on the ground. The whole of these animals died under symptoms of irritant poisoning. The husband also died:—no poison was detected in the stomach, although there were the traces of its action; but on opening the bodies of the animals, the medical witnesses found not only the appearances usually produced by irritant poisons, but arsenic itself was readily discovered in the viscera. This sort of evidence supplied that which was required to complete the case:—for while no poison was detected in the body, no portion of the poisoned soup could be procured. The prisoner was convicted and executed.

Good negative as well as affirmative evidence may be sometimes obtained by the examination of the bodies of animals alleged to have been poisoned. The following case is singular in this respect:—A woman named *Higgins* was tried at the Warwick Summer Assizes, in August 1831, for the murder of her uncle by poisoning him with arsenic. Her guilt was throughout made very clear. It was proved that she had bought arsenic, and when required to account for the possession of the poison, she said that it was for the purpose of destroying vermin—the excuse resorted to by all murderers. She went, however, farther than this; and actually pointed out, in corroboration of her statement, a dead mouse, which she said had been killed by the poison. This turned out to be an unfortunate part of her defence, for the medical witnesses showed that the mouse had not died from the effects of arsenic.

In the above cases, it will be seen that the evidence from the effects of poison was accidental, and ancilary to the main facts of poisoning. There is, however, one instance wherein evidence from experiments on animals cautiously performed, may be of some importance on a criminal trial. I allude to the case where the poisonous substance is not of a nature readily to admit of a chemical analysis, as for example in substances belonging to the narcotic or narcotico-irritant class of poisons. In such a case, if the death of an animal take place under the ordinary symptoms of poisoning from administration of a substance, part of which has been taken by the person whose life was thus attempted, the evidence is very conclusive. This remark applies only to liquids or solids, which are made the vehicle of the poison,—not to any matters vomited or found after death in the stomach. The results here would be fallacious; because such matters may, without containing any poison whatever, give rise to vomiting and other symptoms in an animal. Foderé mentions a case where a young child, after having partaken of some broth, fell into a state of stupor, lost all power of deglutition, and foamed at the mouth. Some of the meat from which the broth was made, was given to a cat. The animal was seized with convulsive fits, alternating with stupor, and died in about five hours. It was rendered probable from the symptoms, as well as from an examination of the body of this animal, that these effects were caused by the introduction of a narcotic plant (hyoscyamus) into the broth. (Méd. Lég. iv. 72.) A remarkable instance of this kind of evidence will be found under PHOSPHORUS, in which a shepherd and his dog were poisoned by this substance, which was detected in the stomach of both after fourteen days' interment. In several instances of late years this kind of evidence has been received in an English court of law. A woman named *Sherrington* was tried at the Liverpool Spring Assizes in 1838, for the attempt to administer poison to one Mary Byres. The evidence showed that the prisoner had sent to the prosecutrix a pudding, by two young children. On the way, these children tasted it, and finding that it had an unpleasant taste, the prosecutrix was put on her guard. The pudding was sent to a surgeon to be analysed; but he could detect no poison in it. He suspected, however, that it contained a vegetable narcotic poison. He gave a piece about the size of an egg to a dog. In twenty minutes the dog became sick—in forty minutes it lost the use of its limbs—and died in three hours. The prisoner was convicted. Cases in which evidence of this kind, accidentally obtained, has been made available on charges of criminal poisoning, are now very numerous. (See *Reg.* v. *Foster*, Suffolk Lent Assizes, 1847.)

Is the flesh of poisoned animals poisonous?—This is a question which it is necessary to consider, because poultry and game are not unfrequently poisoned wilfully or accidentally, and in this state they may be eaten unsuspectingly. It is well known that grain is often saturated with a solution of arsenic for agricultural purposes before it is sown: if this grain be eaten by poultry, it will destroy them; and a question may arise as to the effects which the flesh of the animals so poisoned, is liable to produce on man. In other instances poison has been placed in the way of these animals, with the malicious object of destroying them. Thus oats saturated with arsenic, or with that poison intermixed, have been placed in game-preserves, for the purpose of destroying pheasants and other birds. During the last spring (1846) two blackcocks were sent to me for examination, from the extensive preserves of a nobleman in Scotland. They had been found dead on the grounds. A quantity of arsenic was discovered intermixed with oats and the shoots of the larch, in the crops and gizzards of each bird, and arsenic existed also in the pectoral muscles and soft organs. There had been previously a very large destruction of game on the estate, as it was inferred from poison. There is hardly a doubt that, when the animal dies soon after the ingestion of poison, and obviously from its effects, the flesh would be poisonous to man, although it might require a large quantity of the flesh to produce a fatal result. Dr. Christison reports a case which renders this opinion highly probable. (On Poisons, 81.) This subject has been examined in reference to sheep by M. A. Guérard. (Ann d'Hyg. 1843, i. 468.) Some sheep were poisoned by arsenic, and it became important to determine how far their flesh was rendered poisonous as food. A Commission was appointed by the French Academy to make inquiry on the subject, and M. Guérard has furnished a summary of the results. The sheep submitted to experiment appeared well, even when they were daily passing arsenic through the fæces and urine. On giving to a young dog, the flesh of a sheep which had died from arsenic, the animal after two days was seized with diarrhœa, and arsenic was detected in the fæces and urine. Another dog, which ate the viscera, previously washed, had vomiting and symptoms of a more serious kind; it became thinner, but did not die from the effects of the poison. These results proved that the flesh of poisoned animals is noxious; but if they live sufficiently long, the whole of the arsenic is voided in the urine and fæces, and the flesh may then be eaten with impunity. In an experiment on one sheep, arsenic was found in the fæces twenty-two hours after the introduction of the poison into the stomach. Its elimination was daily traced; and fifteen days after it had ceased to appear in the fæces, it was found in the urine. It ceased to appear in the urine on the thirty-fifth day: and when the animal was killed on the thirty-eighth day, no arsenic was found in its body. Six persons ate of the flesh without suffering any ill effects, and a dog ate the viscera without manifesting any symptoms of poisoning. The flesh, therefore, is only noxious in the early or acute stage of poisoning, and it is not fit for food until three or four days after arsenic has ceased to appear in the urine. Arsenic and corrosive sublimate are much used in this country as a lotion for the purpose of destroying the fly in sheep; but it is not likely that any question will ever arise respecting a poisonous impregnation of the flesh from this source, unless the animals be speedily killed. In a case reported by Mr. Annan, two sheep died from the effects of the external application of corrosive sublimate, a poison which is most easily absorbed. (Med. Times, July 25, 1846, 331.) The flesh of these animals might have proved dangerous if it had been eaten.

The flesh of animals poisoned by copper, has been known to produce serious effects among those who have eaten it as food. Dr. Galtier relates the following case. A pig which had been fed with corn soaked in blue vitriol, was so affected that the owner had it killed, and sold the carcase to a butcher. Seventeen persons who ate of the flesh of this animal were seized with violent colic, and those who ate the blood made into black puddings also suffered severely. The milk of a goat, which had eaten sour food out of a copper vessel, occasioned nausea, vomiting, colic, diarrhœa, cramp, and other alarming symptoms, among fifteen persons who partook of it. They had before taken milk from the same goat without injurious consequences. The animal itself became ill, and died on the third day under all the symptoms of poisoning. The mucous membrane of the small intestines was found inflamed. (Toxicologie, i. 631.) It is to be regretted that no analysis of the food was made in these cases of acute poisoning.

It is a curious fact, that the bodies of animals may, in some instances, be made the vehicles of transferring poison to the human subject, while the animals themselves do not suffer from its effects. Thus the flesh of the pheasant, which feeds on the buds of the *Kalmia latifolia*, in North America, is deemed poisonous during the winter and spring. (Beck's Med. Jur. 854.) The flesh of hares which have fed upon *Rhododendron chrysanthum*, is considered to be poisonous. A singular case occurred in France, in which a whole family near Toulouse was poisoned by a dish of snails. The symptoms under which they suffered were those of narcotico-irritant poisoning; and it was found upon inquiry, that the snails had been gathered from bushes where they had fed upon the leaves and young shoots of the *Coriaria myrtifolia*, a powerful vegetable poison. (Gaz. Médicale, Oct. 1842; also Med. Gaz. xxxi. 237.) It has been long known, that honey, derived from bees which have fed upon the *rhododendron*, *calmia*, *azalea*, or *datura*, acts as a narcotico-irritant poison, producing vomiting, diarrhœa, and vertigo. In the chapter on ANIMAL IRRITANTS some other facts will be mentioned, from which it would appear that the milk of cows fed in certain districts of America is poisonous, and gives rise to serious symptoms, whether taken as milk or made into cheese. The flesh of the animal possesses also poisonous properties; while the animal itself does not suffer in health from feeding on the plants. These facts are explicable on the supposition that there are specific idiosyncrasies among different classes of beings, thus rendering what is innoxious to one, a poison to another.

I have elsewhere (p. 33, ante) objected to certain physiological experiments on animals, as being liable to lead to inconclusive results. All experiments based on the injection of poisons into the blood, are open to the objection that great violence is done to the circulation; and it appears to me that no inferences derived from them can be fairly applied to legal medicine. Any substance thrown into the circulation may thus destroy life by a mechanical action. M. Dupuy has lately come to the conclusion that the *brain* of the sheep kills animals even more rapidly than *corrosive sublimate!* This extraordinary conclusion was derived from an experiment in which a solution of cerebral matter was injected into the crural vein of an animal, and death took place in a few minutes. (Med. Gaz. xxxix. 745.) It is not at all improbable that death was caused under the circumstances, but it is preposterous to compare the effects of sheep's brain with those of corrosive sublimate. Upon similar crude experiments a solution of fæces, or of some most common articles of food, might be inferred to be more poisonous than arsenic!

CHAPTER XIII.

WAS THE DEATH OF A PERSON PRODUCED BY POISON, OR BY ANY OTHER LATENT OR SECONDARY CAUSE? DEATH FROM UNWHOLESOME FOOD—DEATH FROM DISEASE SIMULATING POISONING—CAUTION IN FORMING A DIAGNOSIS—CASES OF SUSPECTED POISONING—DEATH FROM VIOLENCE SUPERVENING ON POISON. QUESTIONS OF HOMICIDE AND SUICIDE—OF TWO POISONOUS SUBSTANCES TAKEN BY THE DECEASED, WHICH CAUSED DEATH?

We have hitherto considered those facts which indicate in a disputed case whether poison has or has not been the *cause of death*, in a previously healthy subject. We have supposed that the question of poisoning would turn simply on the affirmative or negative, and be established or disproved by the medical evidence. We meet with cases, however, in medico-legal practice, wherein the question presents itself under another aspect. Thus poison may have been taken or administered; the fact of poisoning may be established by the symptoms, post mortem-appearances, and the actual discovery of the substance in the food, in the vomited matters, or in the stomach of the deceased after death. All these points may be freely conceded; but the defence will rest upon the question, "Whether or not, the poison so administered, was actually the cause of death." To establish a charge of murder against a prisoner, it must be proved that poison was certainly and indisputably the cause of death. Any proof short of this, as the existence of mere probability, doubt or suspicion, will of course lead to an acquittal. (See the case of *Pouchon*, Ann. d'Hyg. 1844, i. 431.) Thus, then, the medico-legal question would be:—*Was death produced by poison, or by any other latent or secondary cause?* The witness will be required to state which of the two probable or co-existing causes actually destroyed life. It may be remarked, that whenever we obtain those proofs of poisoning which have here been assumed to exist, the presumption is always in favour of death from poison: but it is not the less necessary for a medical jurist to determine, by a careful inspection of all the cavities of the body, whether death might not have been due to some insidious disease. In a case at all involved in doubt, negative evidence is as important as that which is affirmative; and a great error would be in many cases committed, if the examination of a body were stopped so soon as the traces of the action of poison had been discovered. In *Donellan's* case, the head of the deceased, Sir T. Boughton, was not examined, an omission which might, had the general evidence been less clear, have led to difficulty; for the disease from which it was afterwards alleged the symptoms of the deceased might have proceeded (apoplexy and epilepsy) have their seat of morbid changes in that part of the body. An inspection of the head might, it is true, have thrown no light upon the question; but this is not the point—a medical witness must not omit this duty, and then excuse himself by saying that no morbid changes might have been found. The assumption will always be as much against him, as in his favour.

Cases in which the administration of poison is admitted, and death referred to some other cause, although not common in Courts of law, are sufficiently frequent to demand the serious attention of the practitioner. The following appear to me to embrace the chief points on which a defence of this kind may rest.

1. DEATH MAY BE CAUSED BY IMPROPER FOOD.—It has been mentioned in a preceding chapter (ante, p. 21,) that some kinds of food will cause death under symptoms resembling those of irritant poisoning. Such cases are not common, and they appear to depend often on idiosyncrasy or peculiarity of constitution. If poison be taken with such food, we might safely refer death to the former, provided the case took the usual course; and that death was preceded by all or a majority of the characters peculiar to the kind of poison taken. If any of these characters are wanting, this must weaken the evidence; but in most instances, it will be found that the symptoms of acute poisoning are so well marked, as to extinguish those which may have depended upon the unwholesome food. Each case must be judged of by itself; no general rules for a decision can be laid down. Still, it must be remembered, that death is not a very common consequence of unwholesome food, while it is the usual result of an active poison.

2. DEATH MAY BE CAUSED BY DISEASE.—This is a case which more frequently presents itself for our consideration; since poison is often administered to persons while labouring under disease. On a post-mortem examination, we may find, besides indications of poison, marks of extensive disease. When this happens, the chief point to be considered is, whether the disease has advanced to that degree to account for *rapid or sudden death;* for this is one of the essential characters of acute poisoning. Should the history of the case be known, our judgment may be assisted by observing whether the symptoms preceding death were referable to a diseased condition of the body, or to poison. We cannot deny that singular coincidences may occur. A man may have taken irritant poison, and yet death be occasioned by abscesses in the brain, or the lungs,—by sudden hemorrhage, or other causes. If the poison were of a nature to cut short life suddenly, we could not hesitate to refer death to it. Thus it is scarcely possible to admit, when prussic acid is the poison, that death should be referred to some diseased condition of the body found on a post-mortem examination. Whether the person be labouring under illness or not, the taking of this poison would be sufficient to account for death. The only exception would be where the prussic acid was in small quantity, and might have been derived from some accidental source (*Reg.* v. *Tawell*, Bucks Lent Assizes, 1845.) It is not always so easy, however, to determine this question in other cases of poisoning; for whether the substance taken be opium or arsenic, there is time for latent disease of the heart, brain or lungs, to cut short life. The history of the symptoms preceding death, will enable us in general to return an answer. Without this history, or some strong corroborative evidence, a medical opinion can be little more than a conjecture.

Several complex cases of this description have occurred in reference to diseases of the stomach, the persons labouring under such diseases having had poison administered to them. Thus, the organ has been found perforated, and the question has been not so much what caused the perforation, as whether the perforation or the poison caused death. A woman swallowed, by mistake, half an ounce of powdered chloride of barium dissolved in warm water. Nausea and vomiting of a watery mucus supervened, with twitchings of the facial muscles, and convulsive motions of the hands and feet. The symptoms continued to increase in severity, and she died about two hours from the time of taking the poison, under the most violent convulsions. On inspection, the stomach was found perforated posteriorly, in the lesser curvature near the cardiac orifice. The aperture was of an oval form, three lines in diameter externally, and almost twice as large internally. The margin appeared swollen, and the mucous membrane, for about two inches round, was much thickened, and covered with a bloody mucus. The stomach and small intestines were highly inflamed: the cavity of the former contained mucus and coagulated blood. The pharynx and œsophagus presented slight marks of inflammation. The poison was found in the stomach by chemical analysis. Wildberg, who has reported this case, suggested that the perforation was due to the previous disease, and not to the poison taken. This is very probable, for the characters of the aperture were those of perforation from disease; and it would be very unlikely that the chloride of barium, if it led to perforation of the stomach at all, should have given rise to this effect in two hours. It is not stated whether the woman suffered from any symptoms of gastric irritation prior to taking the poison, nor whether the contents of the stomach were found extravasated and the peritoneum inflamed. But there can be no doubt that the woman died from the effects of the poison. This was clearly indicated by the nature of the symptoms and the post-mortem appearances. Admitting that no mistake was made respecting the time at which the poison was swallowed, it must be considered remarkable that this substance should have destroyed life, and left such extensive marks of irritation in the alimentary canal, in the short space of two hours.

The following case was tried at the Taunton Spring Assizes, 1836. A woman named *Edney* was charged with the murder of her husband, by poisoning him with arsenic. It appeared in evidence, that the deceased was attacked with severe pain in the abdomen, and vomiting, shortly after having eaten his dinner, which was prepared for him by the prisoner. Medical assistance was called in; but the man became worse, and he died in sixty hours after the first attack. It was shown that arsenic had been probably given to him at the dinner; and also on several other occasions, when it was supposed to have been substituted for some medicine prescribed for him, his symptoms having been uniformly aggravated after each dose. The chemical evidence was very clear:—arsenic was discovered in the vessel in which the dinner was dressed; also in the stomach of the deceased; and the poison was traced to the possession of the prisoner. On an examination of the body, a scirrhous ulcer, evidently of long standing, was found in the stomach, near the pyloric orifice. It was about the size of a shilling, had a dark appearance, and the margin was inflamed. The mucous membrane of the stomach, as well as the duodenum, was in such a high state of inflammation that it resembled red velvet. The defence on the trial was, that the symptoms and death of the deceased were due to the scirrhous ulcer, and not to poison. It was shown that the deceased had suffered from a gnawing pain of the stomach for a very long period; and it was thought by himself, as well as by others who saw him, that this last attack of illness was nothing more than an aggravation of his old complaint. The medical witness, however, did not hesitate to refer the symptoms and death to arsenic, for the following reasons:—The symptoms occurred suddenly and violently, after a meal at which arsenic was proved to have been administered. Some of these symptoms were peculiar to arsenic, and totally unconnected with the disease under which the deceased was labouring. Pain and vomiting might be ascribed to either cause; but the intense thirst not previously experienced, well-marked inflammation of the conjunctivæ, coldness of the body, and before death paralysis of the extremities with loss of sight, were symptoms unquestionably owing to the operation of arsenic, and not to the effect of chronic disease. This disease was not likely to destroy life with such rapidity and under such severe symptoms. The post-mortem appearances corroborated the opinion founded on the symptoms, and showed that death was really due to an active irritant poison. The woman was convicted upon this evidence.

The more recent case of *Reg.* v. *Foster* (Suffolk Lent Ass. 1847,) also furnishes an important caution to medical witnesses. The prisoner was here charged with poisoning her husband. He had died somewhat suddenly: his body was inspected, and death was referred by the inspectors to an inflamed state of the kidney, and a rupture, to a very small extent of the inferior vena cava. The stomach and intestines were subsequently examined by Mr. Image and Mr. Newham, of Bury St. Edmunds, and a large quantity of arsenic was found. The whole of the tissues were completely impregnated with it. They properly ascribed death to arsenic, considering that the condition of the kidney was not a sufficient cause. The injury to the vena cava was no more than what might have arisen from an accidental puncture during the post-mortem examination. This case is otherwise remarkable from the fact that the prisoner was convicted —although there was no apparent motive for the crime, although no arsenic had been traced to her possession, and there was no direct proof of administration.

The next case occurred in Germany a few years since. A woman, after an illness of many weeks, during which she was subject to constant vomiting and other symptoms of disease in the stomach, died suddenly under suspicious circumstances, and her husband was accused of having poisoned her. The parties had lived unhappily together. The prisoner, under the pretence of relieving her disorder, gave her a white powder and a mixture of *boletus cervinus.* Soon after taking this powder she became much worse, and severe pain in the abdomen with diarrhœa came on. She died nine days after taking the powder! and a physician who saw her shortly before death, considered her to be labouring under the effects of irritant poison. The deceased gave to the physician, the glass from which she had taken the alleged medicine. This contained a white powder, which, on examination, proved to be arsenic. There were well-marked appearances of poisoning in the stomach. The whole of the interior was of a dull reddish brown colour: the lining membrane being in some parts so softened as to have a gelatinous consistency. About half an inch from the pylorous, there was a perforation of the coats of the organ. The edges of the aperture were hard, and had a cicatrized appearance. The stomach contained about twelve ounces of a reddish coloured liquid. The intestines were slightly inflamed. The medicine alleged to have been given by the prisoner to the deceased, was proved to have been arsenic in a decoction of the *boletus cervinus.* The contents of the stomach and intestines yielded no trace of poison, but the analysis does not appear from the report to have been very skilfully conducted. The medical opinion given at the trial was, that the deceased had died from arsenic, and not, as it was alleged, from disease. The prisoner was acquitted of the charge, the Court doubting the correctness of the medical opinion in favour of death from poison. The witnesses were asked, whether they would swear, from the post-mortem appearances *alone*, that the deceased had died from arsenic; but this they declined doing. The previous disease of the stomach, and illness of the deceased, were, in the judgment of the Court, a sufficient cause of the symptoms and death. It is proper to state also, that the evidence of administration by the prisoner was defective. (Rust's Magazin, 1837. 50 B. 2 H.)

I have already referred to a case, in which a woman labouring under malformation of the heart was supposed to have taken nux vomica, and to have died from its effects. The facts, however, showed that death had been really caused by an obstruction to the circulation through the heart (ante, p. 65.)

The two following cases are related by Henke. A young girl died under suspicious circumstances, and an inspection of the body was ordered. The viscera were found healthy, except those of the abdomen. The stomach contained three ounces of a reddish coloured liquid. Its mucous membrane was of a dark red colour, and near the pylorus were several spots of a clear yellow hue. The contents of the stomach, on analysis, yielded arsenic. The account given by the mother was, that the deceased, some weeks before, had met with a fall, after which she complained of pain in her side. Shortly before her death she said she felt ill, and vomited repeatedly,—she went to bed early, and died without being convulsed. The medical opinion was, that she had been poisoned; but the Court held, that the fact of poisoning was not proved, and the prisoner charged with the crime, was acquitted. This case shows that there is great difficulty in forming a medical opinion, when there is no satisfactory account of the symptoms preceding death. In the case of *The Queen* v. *Jennings* (Berks Lent Assizes, 1845,) the deceased was not seen by a medical man during life, but the stomach was inflamed, and arsenic was found in it. The only account of the symptoms preceding death, was derived from the evidence of a young girl. The Court held that the fact of poisoning was established.

In the next case a man was charged with having given to his wife, who had been for a long time ill, a small quantity of arsenic, in four different doses. The only symptoms that followed, were general illness and vomiting. Another and stronger dose was then, it is supposed, administered: and after suffering severe pain, the woman died the day following. The body was inspected twenty-four hours afterwards. In the abdomen, the pancreas was found enlarged and in a scirrhous state, evidently proceeding from chronic disease. The lining membrane of the stomach was inflamed, and it presented gangrenous spots. It contained a greyish coloured liquid having a gritty feel. The uterus was in a state of scirrhous enlargement. The contents of the stomach, on analysis, were found to contain arsenic. The medical opinion was, that notwithstanding the marks of extensive disease in the viscera of the abdomen,—the post-mortem appearances and the detection of the poison in the viscera, proved that the deceased had died from arsenic. The counsel, in defence, raised objections to this view, on the ground that the head had not been examined, and that the chemical analysis was defective. The Faculty of Leipsic being appealed to, overruled the objections. The diseased state of the pancreas might, in their opinion, have given rise to vomiting, emaciation, and death, but not to so sudden a death. The chemical analysis, although in some respects defective, sufficiently demonstrated the presence of arsenic in the viscera. (Zeitschrift der S. A.)

A case of great interest in a medico-legal view has been reported by Dr. Carson. (Monthly Journal of Medical Science, Aug. 1846, p. 142.) A sailor after making a hearty meal was seized with intense pain in the abdomen, and died, without any relief from treatment, in about fifteen hours. On inspection, a large opening was found in the stomach, and the whole of the mucous membrane at the cardiac end was intensely injected. The small intestines, from the stomach to the cæcum, were equally injected, and appeared like red velvet. A number of round worms (*L. teres*) were found in the small intestines, some of them still living. The other organs of the chest and abdomen were healthy. There had been an effusion of fluid into the peritoneum (through the perforation,) but there was no sign of peritonitis. Dr. Brett examined the stomach, its contents, and the fluids of the abdomen; and a trace of arsenic was discovered, so slight that Dr. Brett did not consider it sufficient to enable him to give an opinion that arsenic was the cause of death. The jury returned a verdict, leaving the real cause of death in doubt, *i. e.* that the deceased died from inflammation and perforation of the stomach, which might have been produced by arsenic or natural causes. Dr. Carson considered that the rupture of the stomach was due to the rapid development of gas as a consequence of the acetous fermentation of the contents.

As arsenic was really discovered in this case, it is obvious that it must have been taken by the deceased, although this does not appear from the symptoms and moral circumstances. What was the cause of death—disease or poison? Arsenic does not commonly produce perforation, and it is very unlikely that it should have caused a large aperture in the stomach in fifteen hours. But arsenic would account for the highly inflamed condition of the alimentary canal, and the actual quantity found in the body after death, however small, is of course merely a residue of what has killed. The report states that no effusion of lymph, or other sign of peritonitis, was observed. The perforation, therefore, if the cause of death, does not appear to have operated fatally in the usual way. The aperture in the stomach could not be ascribed to the worms, as these were found in the small intestines. This is a most difficult case on which to express an opinion, but, taking the whole of the facts, with the positive evidence of the presence of arsenic, it appears highly probable that death was caused by poison. The only other view of the matter is, that there was rupture of the stomach operating fatally by shock. This leaves unexplained the extensively inflamed condition of the stomach and intestines.

In the following number of the same journal (Sept. 1846, p. 184) is another case by Dr. Paterson, in which no doubt existed that poison had been taken, but the cause of death appeared somewhat obscure. A girl, æt. 18, swallowed a drachm of King's yellow (a mixture of sulphuret of arsenic, lime, and sulphur.) In about two hours she began to vomit, and she still vomited when admitted into the Infirmary, *i. e.* about ten or twelve hours after having taken the poison. When admitted, there was great anxiety, with collapse, coldness of surface, and a scarcely perceptible pulse. On the day following, the signs of irritation in the alimentary canal abated under treatment, and symptoms of acute bronchitis supervened. She died sixty hours after having taken the poison. Appearances indicative of inflammation were found in the larynx, trachea, bronchi and lungs: but there was no sign of active inflammation about the stomach, and the intestines were healthy throughout. There could be no doubt that death was immediately caused by bronchitis: but the question to decide was, whether this had arisen from the usual accidental causes, or whether it was produced by the poison. From the fact that the mucous membrane of the trachea and lungs has been occasionally found inflamed in cases of arsenical poisoning, Dr. Paterson concluded that the arsenic was here the remote cause, and that the inflammatory action probably extended by continuity from the alimentary canal into the air-passages. This question is of considerable importance. Let us suppose that some person had been charged with criminally administering the poison. Could he have been convicted of murder under the circumstances? Could a medical witness have sworn without hesitation that the poison produced the fatal bronchitis, and caused death; and that it was quite impossible that the disease could have arisen by any coincidence from accidental causes? It appears to me that such an opinion would not have been justifiable, because acute laryngitis, although it may by possibility occur, cannot be reckoned among the ordinary fatal sequelæ of arsenical poisoning; and it would be exceedingly difficult to produce a single instance in which arsenic had clearly and indisputably caused death by exciting inflammation of the mucous membrane of the air-passages.

Cases of this description are very numerous, and the facts are highly instructive. They show how difficult it is, in many instances, for a medical jurist to give a positive opinion whether a person has died from poison or disease. A large number of such cases have been referred to me within the last ten years. A few of the more recent, which appear to illustrate some important principles in reference to the evidence of general poisoning, are subjoined.

Of the first case, I obtained the following history from the surgeon who consulted me on the subject. "I was sent for to see a patient who had been amaurotic and in some degree paralytic for about seven years. He was suffering from general derangement of the system, without any marked symptoms, and expressed himself as having *no pain* either in the head, stomach, or bowels. I prescribed for him an aperient, and some diffusive stimuli. He continued in much the same state for about two days; and on the fourth day was attacked with vomiting of a brownish matter, but unattended with pain. The abdomen was much distended with flatus, and quite tympanitic. On the evening of this day, while sitting on the close-stool, he was seized with faintness to such an extent, as to induce those about him to suppose that he was sinking; but after a short time he rallied. The next day he appeared worse; but still there was no marked symptom of disease, and in the evening he complained of a frequent desire to make water, but he was not able to pass much. He was now seen by a medical friend, who found him in such a hopeless state, that he thought any interference useless. The next morning between nine and ten o'clock he expressed a frequent desire to micturate: he got out of bed, but soon fainted, after which a good deal of urine passed away involuntarily; and in a short time he died. On a *post-mortem examination*, all the organs of the body, except the stomach, liver, kidneys, and bladder, presented a healthy appearance. There was marked *inflammation of the stomach*, which was quite contracted, but free from any signs of ulceration or corrosion. It contained about two drachms of a turbid liquid, possessing no peculiar odour. The vessels of the stomach were much injected, and the lining membrane presented patches of inflammation. The same appearances of redness was met with for about four inches in the upper part of the duodenum; but this almost entirely disappeared after the third day. The intestines contained no solid nor liquid matter, but were distended with flatus. The liver and kidneys were congested, and the bladder contained about two pints of urine. There was no sign of inflammation in the organ, nor was there any appearance of peritonitis. It could not be ascertained that any of the symptoms had been increased after any meal or the administration of food, nor was it possible to account for the gastritis, of which during life the deceased had no symptom, except slight vomiting. A coroner's inquest was held in this case, and a verdict was returned of "Died from inflammation of the stomach;" but the cause of the inflammation could not be assigned." *After* the inquest it was considered advisable to examine the stomach, and the liquid taken from it. The stomach and its contents were forwarded to me for that purpose. The liquid was of a brownish colour, slightly acid, and partially decomposed. It contained a turbid brownish sediment, and the whole amounted to two drachms. The liquid and the sediment were separately examined for arsenic and the more common irritant poisons, but not a trace of any of these bodies could be detected in them. The mucus scraped from the inflamed stomach was then examined, but with a like result. The whole of the organ was then cut into small pieces, and boiled in muriatic acid and water for two hours. The liquid thus obtained was of a dark colour, highly acid, clear, and free from viscidity. It amounted in quantity to about four ounces. A piece of bright copper was introduced into the liquid, when it had been brought to the boiling point,—and allowed to remain for half an hour. For some minutes the copper underwent no change; but in less than a quarter of an hour the whole surface of the metal acquired a dark grey tarnish, very closely resembling that produced by arsenic under the same circumstances. The copper was dried, and the grey powder scraped from the surface and heated in a reduction tube, but no white crystalline sublimate could be obtained. It should be observed, that the greater part of the powder scraped off was metallic copper, the coating amounting to only a very thin film. The result was therefore by no means conclusive; and as neither the liver, spleen, nor any part of the body of the deceased could be procured, in order to confirm or remove the suspicion excited by this deposit on the metal, the investigation was abandoned. The symptoms, so far as they could be observed in the case, were not those of poisoning by arsenic; and yet it is difficult to account for the well-marked inflammation of the stomach and duodenum, and the chemical effect on the copper; for the whole of the materials employed in the analysis were pure. It was proposed that the body of the deceased should be disinterred for the examination of the liver and other viscera; but the coroner did not consider that the circumstances warranted this step, as there was no moral evidence that any party had administered poison to the deceased.

An investigation of some importance took place before Mr. Lewis, coroner for Essex, in October 1846, in which a woman, already committed for the murder of her two children by poison, was implicated. The accused had paid several visits to the mother of the deceased, an infant aged ten months, and had, as it was supposed, on more than one occasion put some substance into its mouth, which according to the deposition of the witnesses, was of a pink colour. Immediately after this it is stated the child appeared very ill, was sick, dribbled or was salivated, its jaw dropped, and it appeared convulsed; on one occasion it shortly fell fast asleep. The last time that the accused had had access to the child, and was seen to give it any thing, was about *three months* before its death. It was stated that the child had been healthy up to this period, but it then fell off, was liable to attacks of vomiting after food: it gradually wasted away, and died. A careful inspection of the body was made; the viscera were in a healthy condition, except those of the abdomen. The mesenteric glands were considerably enlarged. The greater end of the stomach was so softened, that it gave way on being touched: some fluid escaped, which was collected for analysis. The gall-bladder was unusually distended with bile. The liquid of the stomach, with the viscera, were forwarded to me for examination. The liquid was of a vinous nature (some wine had been given to the child before death,) mixed with sugar and partially digested food: there was no trace of any kind of poison either in it or in the tissues of the stomach and intestines. The opinion given was, that, so far as the examination went, the child might have died from natural causes, *i. e.* from disease of the mesenteric glands; and a verdict was returned accordingly.

The circumstances of this case were of an exceedingly suspicious kind. The accused had a bad character: she was already committed on a charge of destroying by poison two of her own children eighteen months before. Her house had been searched, and no less than eighteen different medicinal preparations, some of them active poisons, were found. The whole of these had been sent to me for analysis by the Essex magistrates; and there could be no doubt from the facts proved at the inquest, that some substance had been given to the child by this woman, who had so suspiciously sought it out, and endeavoured to get it from the keeping of its mother. The alleged motive for this act need not be entered into: the child was illegitimate, and this part of the case implicated another party. Here, then, much caution was required in giving an opinion, for the moral evidence was exceedingly strong.

The poisons found in the possession of this woman were compounds of lead, mercury (including the ointment of the nitrate of this metal,) and cantharides. A sugar-plum, which was supposed to contain arsenic, was proved to consist of nothing more than sugar and starch. The only substance of a pink colour found in her possession, was some sulphur-ointment coloured red by a small quantity of cinnabar. The main question was, whether a dose of any of these substances, given *three months before*, could have caused the symptoms and subsequent death. The symptoms could hardly be reconciled with the known effects of any of these substances: the dribbling even is stated to have come on *immediately*, and does not appear to have continued; besides, this might be referred to commencing dentition. It was represented to the coroner, that if the dose of the mercurial preparation was small, the symptoms would have soon passed away, and the child would have recovered: if large, they would have been of a more intense kind, would have progressively increased until death, and this would have happened probably within a few days or a fortnight of the time at which the poison was administered. Not one of the eighteen substances (given in a single dose,) which were found in the possession of the accused, and analysed, could account for the lingering and death of this child three months after the period of administration. Considering that no mineral poison was detected in the contents or tissues of the stomach and intestines, and that there was not the least appearance of the past or recent action of any poison in or about the alimentary canal (the softening of the stomach being a post-mortem change independent of poison,) there was an entire want of evidence to show that the deceased had died from poison. On the other hand, the slow emaciation of the body, with the great enlargement of the mesenteric glands, rendered it probable that there was here sufficient to account for death from natural causes. It was, however, a question whether this enlargement of the glands might not have been a secondary result of some noxious substance administered three months before death. Any cause of irritation in the alimentary canal might probably lead to the enlargement of the glands; but such an appearance has not, so far as I know, ever been witnessed as an effect either of acute or chronic irritant poisoning. When, moreover, it is considered that the enlargement of these glands is one of the most common forms of natural disease among the children of the poor, and is a very frequent cause of death,—also that the causes of it—scrofulous constitution, improper food, and obstructed dentition—are very common, it would be improper to refer this morbid condition, in the absence of direct proof, to the action of a mineral poison. There was no doubt that some substance had been given to the child, and perhaps with improper intention; but there was an entire want of proof that this substance had been the cause of death three months afterwards. The conclusions from the whole of the medical facts were—1. That whatever caused the symptoms, death was not due to poison. 2. That tabes mesenterica, most probably depending on natural causes, was the immediate cause of death. The prisoner was therefore exculpated on this charge.

A child labouring under mesenteric disease may, however, have poison administered to it, and die from its effects. The fact must then rest upon the symptoms, post-mortem appearances, or chemical analysis. The following case was referred to me in June 1846. In this instance death was ascribed, upon insufficient evidence, to mesenteric disease, when it was really due to poison. It also teaches a caution, namely, that in an unknown case, a medical opinion of the cause of death should never be given until an inspection of the body, and an analysis of the contents of the viscera, has been made.

On Monday, June 8th, about ten o'clock P. M., a wicker basket was found in a doorway, containing the body of a male child, apparently about four years old, recently dead. At the coroner's inquest the evidence given by the medical witness was to the effect, "that the deceased child was two years and a half old; that he had never walked; that he was diseased from birth by rickets, and an affection of the mesenteric glands: consequently, there could be no motive for destroying a life which was likely to be cut off by disease." The witness was asked by the coroner if he had made a post-mortem examination of the body. He said he had not. The jury being dissatisfied with this medical opinion, the inquest was adjourned, and another medical gentleman was requested to examine the body, which took place five days after it had been found. The *external appearance* indicated a boy between four and five years of age; light sandy hair, rough and wiry; forehead freckled, as if he had been accustomed to run about the country; head well formed, and the bones all united; full complement of teeth. Trunk and arms well formed; lower extremities rather small; the soles of the feet thick and hard, as though he had been accustomed to make the natural use of them. The *mouth* appeared to have had some corrosive substance applied to it, for the tongue was hard, black, and charred, which appearance extended until it was gradually lost in the pharynx. *The stomach in situ:* The short curvature was very dark, approaching to sloughing; and when secured by ligatures at each orifice for the purpose of removal, this dark portion gave way, and a part of the contents escaped by two or three perforations. The remainder, when emptied into a vessel, looked like thin gruel mixed with bread. This pultaceous mass, diluted with distilled water and filtered, after being submitted to various tests, gave unequivocal evidence of the presence of arsenic.

The gentleman who furnished me with the foregoing particulars brought a portion of the contents of the stomach to me, in order to have an opinion respecting the presence of arsenic. The poison was held dissolved in the fluid in large quantity, and it was obtained in a few minutes, by Reinsch's process, in the state of arsenious acid. But for the dissatisfaction expressed by the jury respecting the first medical opinion of the cause of death before an inspection was made, the usual verdict of "found dead" might have been returned, and the crime of murder by poison have thus remained for ever concealed.

The longer life is protracted after the supposed administration of poison, the more difficult becomes the decision. It will be seen from the facts above related, that a question of this kind can only be satisfactorily settled, by a reference to the particulars attending each case. The following which occurred to Dr. Christison, is in this respect interesting. A man named *Munn* was tried at the Inverary Spring circuit, for the double crime of procuring abortion, and of murder by poisoning. The moral evidence and symptoms together, left no doubt that arsenic had been given; and that the deceased, a girl with whom the prisoner cohabited, had laboured under the effects of this poison in a very aggravated and complex form for twelve days. After that she began to recover rapidly, and in the course of a fortnight was free from every symptom, except weakness and pains in the hands and feet:—in short all things considered, she was thought to be out of danger. But she then became affected with headach and sleeplessness, and died in nineteen days more under symptoms of obscure general fever, without any local inflammation. The medical opinion given was, that granting the girl's first illness, as it appeared from moral and medical evidence, to be owing to arsenic, her death could not be ascribed to that poison with any certainty. (On Poisons, p. 56.)

This question may sometimes present itself to a medical witness under another form, namely, whether a person has died from a medicine exhibited in an improper dose or from disease. Thus a person enfeebled by age or disease, may be killed by a powerful drastic purgative. Infants may be killed by small doses of opium or calomel. Several lives have been already lost by the effects of repeated doses of gamboge and aloes, exhibited in large quantities to enfeebled persons, under the form of Morison's pills; and convictions for manslaughter have taken place on this ground. The questions here will be, 1. Whether the medicine or the disease caused death: or, 2. Whether the medicine accelerated death, by aggravating the disease or rendering it fatal. The responsibility of the accused will depend upon the answers; and it will be for a jury to consider whether there were sufficient knowledge and caution employed by the person prescribing it. The most simple remedies, improperly used, may thus act like poisons, and destroy life. Such cases are commonly too well marked, to admit of much difficulty in deciding as to the real cause of death.

3. Death may have been caused by violence and not by poison.—A person who has taken poison may be maltreated, and the question will arise, whether the poison or the maltreatment was the cause of death. The solution of this question cannot in general be very difficult, when the history of the case is before us. Two instances of this kind are recorded by Christison, both of them quoted from foreign authorities. He suggests, what is highly probable, that their real nature would not have been discovered in this country, owing to the very superficial way in which inquiries into the causes of death are here conducted:—the rule being, not to call for a post-mortem examination of a body unless there be suspicion, when in point of fact, in numerous instances, the inspection may be the only source from which suspicion of violence will proceed; and the very circumstance of holding an inquest impliest something like suspicion as to the cause of death.

Wildberg was called upon to examine the body of a girl, who died while her father was chastising her for stealing. It was supposed by all, that the girl had died from the effects of the violence. On the arms, shoulders, and back, many marks of violent treatment were found, and under some of them blood was extravasated in large quantity. The injuries, although severe, did not appear sufficient to account for the sudden death; he therefore proceeded to examine the cavities, and on opening the stomach he found it very much inflamed, and lined with a white powder, which was proved to be arsenic. It turned out that on the theft being detected, the girl had taken arsenic for fear of her father's anger: she vomited during the flogging, and died in slight convulsions. Upon this Wildberg imputed death to the arsenic, and the man was exculpated.

A woman at Berlin, who had lived on bad terms with her husband, went to bed in perfect health, but soon afterwards her mother found her breathing very hard, and on inquiring into the cause, discovered a wound on the left side of the breast. A surgeon was sent for, and the hæmorrhage, which was slight, was arrested: but the woman died towards morning. On an inspection, it was found that the wound had penetrated the pericardium without touching the heart, and that the hæmorrhage had been caused by a division of one of the intercostal arteries: but very little blood was effused in the chest. Coupling these circumstances with the trifling loss of blood during life, and the fact that she had much vomiting and some convulsions immediately before death, it appeared to the medical examiner that she could not have died from the wound. On a further inspection of the body, signs of corrosion and irritation were found in the mouth, throat, and stomach, and the remains of some nitric acid were discovered in a glass in the room. The facts proved that she had died from poison. (On Poisons, p. 48.) The cause of death may be easily assigned in such cases when the circumstances are known; but it is evident that without great care in conducting post-mortem examinations, the apparent may be sometimes mistaken for the real cause.

The kind of violence may sometimes sufficiently account for death without reference to the poison which may have been taken. The following remarkable case occurred in 1836. A young man was found hanging in his bed-room, quite dead. He was suspended by his cravat, and his feet were within an inch of the floor. The door of the room was fastened on the inside, and it was proved that no one could have had access to it. An earthen pan was found near the bed, containing about a pint of blood, which appeared to have proceeded from a very deep incision in the bend of the left arm of the deceased. The razor with which this had been inflicted, was found on the mantelpiece. It came out in evidence, that on the night previously, the deceased had swallowed a quantity of arsenic, and had suffered severely from the effects of the poison; although, at the time, it was supposed that his illness was due to other causes. In this case there were three modes by which suicide was attempted. The deceased had first taken poison, then wounded, and afterwards hung himself. There could be no doubt that death was caused by hanging; and had the wound been inflicted, and the poison administered by other parties, this opinion might have been safely expressed. Had the body been found hanging in a suspicious locality, these circumstances might have created a strong presumption of murder.

A singular case is reported by M. Desgranges, in which a man was found lying on the ground in an insensible state, with marks of contusions on his body as if he had fallen from the window of his room. He died without recovering his senses. On inspection, a large quantity of carbonate of copper was found diffused through the whole of the alimentary canal. From the facts which came out, the cause of death was referred to this poison. (Med. Gaz. xxxi. 495.)

The real cause of death may not, however, be always so clear; for a severe wound, sufficient to account for death, may have been inflicted on the individual who has taken poison. A case occurred to Mr. Watson, of Edinburgh, which may serve as an illustration. A stout, corpulent woman, aged 60, was brought into the Royal Infirmary, on the evening of the 7th February, 1838, having her throat extensively cut; she died shortly after admission. It was ascertained that she had swallowed two ounces of sulphuric acid, a quarter of an hour before cutting her throat. After having taken the acid, she was seen writhing in great pain: she had then put a razor into her pocket, and left the house to cut her throat. She inflicted the incisions on arriving in the street, was immediately seen, and conveyed to the Infirmary, which was close by. She died in about half an hour after taking the sulphuric acid. (Ed. M. & S. J., April, 1840.) The wound in the throat was very deep, and besides other vessels, it divided completely the internal jugular vein on the left side. At the inspection, there being then no suspicion of poison, it was supposed that the hæmorrhage from the wound sufficiently accounted for death. On opening the abdomen, three-fourths of the stomach were wanting, its coats having been dissolved and decomposed by the action, as it was proved, of strong sulphuric acid. Whether this or the hæmorrhage was the cause of death, it was rather difficult to say: but probably the loss of blood, by weakening the system, accelerated the effect of the shock produced from the extensive disorganization of the stomach by a corrosive poison. Thus both causes may have operated, since it is unusual for sulphuric acid to destroy life within so short a period of time. I need hardly observe, that had the wound been inflicted by another, a most important question would have arisen respecting the degree of criminality to be attached to the party who had inflicted it. For some interesting cases and good practical suggestions on this subject, see Belloc, Cours de Méd. Lég. 148.

4. OF TWO POISONOUS SUBSTANCES TAKEN BY THE DECEASED, WHICH CAUSED DEATH.—This question does not relate so much to the subject of compound poisoning, as to cases of the following kind, which may require careful medical investigation. A person may have poison administered to him while

labouring under the effects of powerful medicine, or some other poison. Thus a patient, while under a course of mercury, may have had corrosive sublimate administered to him with intent to murder. After a certain period, violent salivation with sloughing may ensue, and the patient die. Is death in such a case to be ascribed to the corrosive sublimate, or to the mercurial medicine previously administered? It may be necessary to state, that death is sometimes a result of the severe salivation, induced by the preparations of this metal, prescribed medicinally, in a mild form and in small doses.

In *Butterfield's* case, tried at Croydon many years ago, this question incidentally arose. The prisoner was indicted for administering corrosive sublimate to the deceased. The immediate cause of death was profuse salivation: and this was referred, by the medical witnesses, to the operation of the poison. It was proved, however, in the defence, that about two months previously to this attack, the deceased had been under treatment with some quack-medicine, by which he was violently salivated; but this salivation had entirely ceased, and during the whole of the above-mentioned period he had abstained from taking any mercurial preparation. It was at this time that the corrosive sublimate was supposed to have been secretly administered to him in small doses. The prisoner, however, was acquitted of the charge, on the ground that, as mercury had been introduced into the system of the deceased by the quack-medicine, the fatal salivation might have proceeded from a recurrent operation of this medicine, and not from the poison.

Sometimes the action of the two substances will be so entirely different, as to create no difficulty in determining which caused death. At the Oxford Spring Assizes, 1836, a woman named *Tarver* was indicted for the murder of her husband by poisoning him with arsenic. The evidence showed that the deceased, who was a labourer, went to his work between four and five o'clock in the morning. In about ten minutes afterwards, he became very sick: he continued to become worse until about two o'clock on the same day, when he died, nine hours after his first seizure. He suffered chiefly from a burning heat in the stomach and violent vomiting. Arsenic was traced to the possession of the prisoner. It was proved that she had prepared breakfast for her husband on the morning on which he was seized; and that among other dishes she gave him some rice-pudding. On opening his body, the stomach was found highly inflamed: its surface covered with a grey sanguineous mucus, and at the pyloric extremity there was a large patch of a deep mulberry colour. An analysis of the contents of the organ was made, and arsenic was discovered in them in very large quantity. The defence chiefly rested upon the following point. It was proved that *two days* before the deceased was attacked with this severe illness, he had, of his own accord, taken some pills made of scorched wood-laurel, nitre, and flour; and it was stated by the medical witness that wood-laurel was an acrid poison. He thought, however, that if this substance had acted as such, and had caused death, it would have begun to operate in ten or twelve hours at the farthest. But as the deceased was proved to have been quite well on the whole day after he took these pills, and that forty-eight hours had elapsed before any alarming symptoms began, the witness thought that they could have had no connexion with the symptoms or death of the deceased. The symptoms, as well as the post-mortem appearances, proved that death had been caused by arsenic. The prisoner upon this evidence was convicted, and confessed before execution, that she had put arsenic into the pudding served for her husband's breakfast. The correctness of the medical opinion in this case cannot be disputed. The operation of wood-laurel as a poison, even had it been administered in sufficient quantity to do mischief (a fact not proved in evidence,) was not likely to be suspended for forty-eight hours, and then burst out with such severe symptoms, and destroy life in nine hours.

The following case is of some interest in relation to this question. A woman was found dead, and near her body was a glass containing some sulphuric acid. This gave rise to a suspicion of poisoning. On inspection, the mouth and fauces were covered with a black mucous matter, and the œsophagus was filled with a tarry-looking mass, which had a strong acid reaction. The coats were softened, and the lining membrane was easily detached. The stomach was throughout inflamed, and of a brownish black colour: its coats were so softened, that it could not be moved without lacerating it. It contained a large quantity of a black viscid liquid, in which, as well as in that of the œsophagus, arsenic and sulphuric acid were detected. (Von Raimann, Med. Jahrb. 20 B. 2 S. 221.) Admitting that two poisons were taken in this case, which was taken first? Most probably the arsenic. It is more difficult to say which caused death, because the deceased was not seen during life; and probably she was already labouring under the effects of arsenic when she swallowed the sulphuric acid. The fact of this last poison having been taken, appears to show that it was a case of suicide. But perhaps the sulphuric acid itself contained arsenic as an impurity, since some specimens prepared from arsenical pyrites, are found to be impregnated with a large quantity of this substance.

It is obvious that for the proper investigation of cases of this description, the medical witness should be prepared with a full knowledge of the peculiar properties of most poisons,—the doses in which they prove fatal,—the power which they have of modifying each other's effects,—and the period of time within which they produce their symptoms and commonly destroy life.

CHAPTER XIV.

CONCLUDING REMARKS ON GENERAL POISONING—MORAL AND CIRCUMSTANTIAL PROOFS—LEGAL RELATIONS OF THE SUBJECT—STATISTICS OF POISONING—CORONERS' RETURN OF DEATHS FROM POISON IN 1837-8—RETURN FOR 1840—DEATHS FROM POISON IN FRANCE AND DENMARK.

THE duty of a medical witness as such, is accomplished when he has proved on a charge of criminal poisoning, that death was *certainly* due to poison. The moral and circumstantial evidence must prove that the accused was the party who gave it;—this proof often fails,—the fact of administration cannot be brought home to the accused, and the case falls to the ground. It is not within the province of this work to treat of moral and circumstantial evidence in cases of poisoning. Proofs of this kind, it is true, are sometimes very closely mixed up with the evidence of professional witnesses, and in the foregoing chapters some of these have been already adverted to. A witness must, however, be cautious not to base his opinion, in questions of poisoning, on moral and circumstantial proofs. He is called upon to give a medical opinion of the cause of death, and from *medical* facts only. The moral and circumstantial proofs refer chiefly to the *administration* of poison by a particular party, and the *intent* of the person charged with the crime;—it is therefore considered to fall within the province of the jury alone to decide on their relevancy and value, although it must be confessed, that many of these facts can only be properly estimated by persons versed in medical science. Supposing death by poisoning to have been clearly proved, it may be necessary to ascertain whether the act was the result of *accident*, *suicide*, or *homicide*. This is a question also for a jury to determine, and not for a witness; although its solution often depends upon a proper appreciation of medical circumstances. Suicide or murder will sometimes be inferred, according to the medical evidence given of the effects of certain poisons. Some speedily annihilate volition and the power of locomotion, and therefore render it a question of serious difficulty, whether particular acts could or could not have been performed after the deceased had taken the poison. On the answer to this, may depend the acquittal or conviction of a person charged with the crime.

There is one peculiarity in the legal consequences of the act of killing by poison, namely, that the act itself is generally considered in law to be evidence of malice. If a poison is knowingly administered to another, with the intention of destroying life, the crime is never reduced to manslaughter,—whatever may have been the provocation which the party administering, has received from the person whose life he has thus taken. It is not necessary, therefore, that any particular enmity should be proved to have existed between the prisoner and deceased, although this often weighs as a strong moral circumstance against the former; and the absence of any apparent motive for this crime, on the other hand, is always regarded as a strong presumption in favour of the accused. When a man is killed by a wound in a quarrel, the law will sometimes find an excuse for the act, from the heat and passionate excitement under which the aggressor was labouring at the time; but if the aggressor should avenge himself by secretly administering poison to his adversary, there is no excuse for the act, since it evinces cool, reflecting, and deep-rooted malice. That death by poison should ever amount to manslaughter, therefore, it must be shown, that the substance was administered to or laid in the way of the deceased by mistake, or with innocent intention; and the proof of this always lies with the accused—the law inferring that malice exists until the contrary appears from the evidence. Whether malice exist or not, is however, in general soon made apparent from the evidence of the prosecution.

There is one curious point in our law, namely, that in order to make the killing murder, it is necessary that the party should die from the effects of the poison, within *a year and a day* from the time at which it was taken. (Archbold's Criminal Pleading, 345.) In practice, this restriction is of little importance, because most cases of criminal poisoning, if they prove fatal at all, destroy life within a period much shorter than that required to constitute murder by the law. Still, such a rule as this ought not to be allowed to exist in any shape; because it is quite possible that death may take place from the indirect effects of poisoning (as in the mineral acids,) long after the period assigned. In this respect the laws of Scotland and France seem much more consistent with reason and justice. According to these, a prisoner may be held responsible, at whatever period death may ensue, provided it be clearly traced to the action of the poison.

The present state of the English law with regard to the crime of poisoning is as follows:—

The wilful administration of poison, followed by death, constitutes murder, and capital punishment is annexed to the crime by the common law of England.

Under a recent statute, 1 Vict. c. lxxxv. s. 2, the administration of poison with *intent* to murder, if followed by *bodily injury dangerous to life*, though not by death, is also a capital felony.

By s. 3, of the same statute, the attempt to administer poison with intent to commit murder, although no bodily injury whatever ensue, is an offence liable to be punished by transportation or imprisonment.

[In many of the States from having been British colonies, the common law of England, as it existed at an epoch of their separation from the mother country, is still in force, though more or less modified by subsequent enactments of their respective legislatures. Thus, whilst in some of the States, the same rule holds good as in England, as respects the period during which a death by poison would be regarded as a murder; in others, there being a duly enacted code of penal law, in which there is no recognition of the time within which a murder by poison is punishable as a capital crime; a prisoner is held responsible for the poisoning, at whatever period the death may occur, as in Scotland and France, if it can be shown that the said death was from the effects of the poison. In all the States an attempt to poison, though not followed by death is ranked among the higher crimes, in most of them under the common law, but in others, as in New York, by special enactment.—G.]

In concluding this chapter, I wish to call the attention of the reader to some facts connected with the *statistics* of poisoning. In relation to medico-legal practice, this is a subject of some interest; because it will indicate to the medical jurist, the poisons which are most frequently selected for the purposes of suicide and murder, and with the properties of which, it will be expected that he should be acquainted. Unfortunately, very few tables of this kind have been published; and those which have appeared are defective in many points. One of the best is that which was published some years since from the returns made by the coroners of England, of the number of inquisitions held in the years 1837 and 1838, wherein death was caused by poison. The following is an abstract of the paper, which appeared in the Medical Gazette for November, 1839.

The number of deaths by poison (returned) in the two years above mentioned, were, exclusive of two cases of suffocation by gases, 541, of which number 282 were males, and 252 females. The substances which caused death, may be taken in the following numerical order.

Opium	Laudanum . .	133			
	Opium . . .	42			
	Other preparations .	21	196		
Arsenic			185		
Sulphuric acid			32		
Prussic acid			27		
Oxalic acid			19		
Corrosive sublimate and mercury . .			15		
Mixed or compound poisoning . . .			14		
Oil of bitter almonds			4		
Poisonous mushrooms			4		
Colchicum, nux vomica (of each 3) . .			6		
Nitric acid, caustic alkali, tartar emetic, acet. Morphia, strychnia, deadly nightshade, aconite (of each 2)			14		
Bichrom. potash, nit. silver, Goulard's extract, sulph. iron, mur. tin, hellebore, castor-oil seeds, savin, hemlock, cantharides, cayenne pepper, (of each 1) . .			11		
				527	
Unknown				14	
					541

[A little discrepancy exists in the relative number, probably owing to the fact that in several instances some of the poisons were taken in a compound state. The reader will find the details in the Med. Gaz. xxv. p. 204.]

It will be seen by this table, that the largest proportion of cases of poisoning in England, are those by opium and arsenic: the greater number of the former, being cases of suicide and accident, and of the latter, cases of criminal poisoning. There can be no doubt that the number of deaths from poison which annually occur in England and Wales, are much greater than this table represents. The annual registration of deaths, although defective with respect to the number from individual poisons,—two only, *i. e.* arsenic and opium, being commonly recorded, and these imperfectly,—shows that the mortality from this cause, including overdoses of medicine, is greater than is commonly supposed. I here subjoin a table of the deaths from poison in 1840, drawn up from the sixth Annual Report of the Registrar-General (1844.) The deaths from this cause in 1840 are stated to have been 349, of which number there were 181 males and 168 females. The cases of suicide from poison were 161, being 87 females to 74 males,—the cases of accident or homicide were 188, being 107 males to 81 females. Of the 75 cases of poisoning by opium, 42 occurred in children under five years of age,—a lamentable proof of the extensive mortality among children from the improper administration of this drug. These cases occur among the returned deaths from opium; but under the head of medicines improperly administered, three-fourths of the deaths took place among children under five years of age!

Opium	75
Arsenic	32
Other poisons, including medicines improperly administered	242
Total deaths from poison in 1840	349

It would be a considerable benefit to medical science, if the poisons which caused death, were more distinctly specified in the Registration returns. The poison or medicine should be stated as distinctly as in the returns now made with respect to fatal diseases. The cases of poisoning by arsenic and opium in the above table, are evidently understated.

The two next are tables, the one of the statistics of poisoning in Denmark from the year 1830 to 1835 (Med. Gaz. xxv. 575;) the other of 94 cases observed in France during a period of seven years, 1825-32 (Briand, p. 434.)

IN DENMARK.

Sulphuric or nitric acid, generally diluted	74	
Arsenic	16	
Caustic alkalies	5	
Opium	2	
Litharge, verdigris (of each 1)	2	
		99

IN FRANCE.

Arsenic	60
Fly powder	3
Verdigris	7
Corrosive sublimate	5
Cantharides	5
Nux vomica	4
Nitric acid	2
Acet. lead, carb. lead, sulph. zinc, tartar emetic, opium, prussic acid, mercurial ointment, orpiment (of each 1)	8
	94

It is difficult to compare these two tables with the preceding,—the number of observations being much fewer. Poisoning by arsenic is, however, proved by them to be very common. Out of 616 charges of poisoning in France during a period of twenty years, one-half, if not two-thirds, were cases of poisoning by arsenic. (Flandin, Des Poisons, i. 448.) It is remarkable that the mineral acids should have caused so many deaths in the kingdom of Denmark, the proportion being no less than three-fourths of the whole number.

IRRITANT POISONS.

NON-METALLIC IRRITANTS.

CHAPTER XV.

DIVISION OF IRRITANT POISONS. SULPHURIC ACID OR OIL OF VITRIOL. GENERAL REMARKS. SYMPTOMS CAUSED BY THIS POISON—SOMETIMES PRODUCES SUFFOCATION—ACTION OF THE DILUTED ACID—TIME AT WHICH THE SYMPTOMS BEGIN. CAN THEY CEASE AND REAPPEAR?—POST-MORTEM APPEARANCES IN ACUTE AND CHRONIC CASES. QUANTITY OF ACID REQUIRED TO DESTROY LIFE —FATAL DOSES—PERIOD AT WHICH DEATH TAKES PLACE—TREATMENT—CHEMICAL ANALYSIS—CARBONIZATION OF ORGANIC MATTER BY THE ACID—MODE OF DETECTING THE POISON IN PURE AND MIXED LIQUIDS—OBJECTIONS TO THE TESTS—THE ACID NOT ALWAYS FOUND IN THE STOMACH—ITS DETECTION IN ARTICLES OF CLOTHING—AROMATIC SULPHURIC ACID—SULPHATE OF INDIGO—QUANTITATIVE ANALYSIS.

General remarks.—IRRITANT POISONS may be divided into four groups—the non-metallic—the metalloids—the metallic—and those of an organic nature, *i. e.* derived from the vegetable and animal kingdoms. The non-metallic irritants comprise the mineral acids, oxalic acid, the alkalies, and their salts. According to strict chemical views, the alkalies and their salts should be placed among the metallic irritants; but it will be in many respects, convenient to consider them in the same group with the acids. Besides, although they certainly have metallic bases, the demonstration of the existence of the metal, is never required at the hands of a medical jurist, as in the case of the true metallic irritants. Among the mineral acids, we shall first speak of poisoning by sulphuric acid.

SULPHURIC ACID, OR OIL OF VITRIOL.

This is met with in commerce in two states, either concentrated or diluted. The concentrated acid is a heavy oily-looking liquid, often of a brown colour: it has a strong sharp acid taste—it powerfully reddens vegetable colours, and corrodes and destroys most kinds of organic matter. It is very frequently taken as a poison by suicides; but probably there is no case in which the sufferings of the individual before death are more intense. In medico-legal practice, it is not very common to find that this acid is employed for the purpose of murder. Young children have, however, been destroyed by a quantity of it being poured down the throat; and it is obvious, that a person who is drunk or asleep, may be thus easily killed. With these exceptions, which are of rare occurrence, instances of fatal poisoning by sulphuric acid, may be pretty equally divided into cases of suicide and accident. The taking of this liquid is a very frequent form of self-destruction among females;—less frequent among males, and by no means uncommon as an accident among young children of both sexes. On the discovery of a dead body, poisoned by sulphuric acid, a medical jurist will have, then, especially to consider the age of the deceased. If it be a new-born child, or a very young infant, it is certain that the poison has been homicidally or accidentally administered; if a child, all other circumstances being equal, that it has been swallowed by accident; if an adult, that it has been voluntarily taken for the purposes of suicide. It is to be observed, that there is no poison which can be obtained more readily or without exciting less suspicion than sulphuric acid, since it is used for so many domestic purposes. The only probable case of murder by this poison in an adult, would be where the person was either intoxicated or asleep when it was administered; but even then the individual would be immediately roused. It is not easy to imagine that a criminal, who wished to destroy the life of another, would attempt this by causing him to swallow forcibly a quantity of oil of vitriol, when there are so many other more ready, secret, and speedy means of destruction at hand. It is also impossible that such a substance as this should, like arsenic, be secretly administered in articles of food. Its powerfully acid taste in the smallest quantity, and the fact that the physical qualities of the food would be entirely changed, would certainly lead to a discovery and frustrate the attempt. There are but few instances in which such an attempt to poison, has been made. In one of these, a boy being offended with his mistress, put a quantity of common diluted vitriol into a cup of tea, which she was about to drink. The taste of the tea immediately led to the discovery of the attempt.

SYMPTOMS.

THE CONCENTRATED ACID. When this poison is swallowed in a concentrated form, the symptoms produced, came on *immediately* or during the act of swallowing:—it is one of the most powerful corrosives. There is violent burning pain extending through the fauces and œsophagus to the stomach—the pain is often so severe, that the body is bent. There is an escape of gaseous and frothy matter, followed by retching and vomiting, the latter accompanied by the discharge of shreds of tough mucus and of a liquid of a dark coffee-ground colour, mixed with blood. The mouth is excoriated, the lining membrane and surface of the tongue white or resembling soaked parchment—in one instance the appearance of the mouth was as if it had been smeared with white paint: after a time, the membrane acquires a grey or brownish colour; the cavity is filled with a thick viscid sputa, rendering speaking and deglutition very difficult. If the poison has been administered by a spoon, or the phial containing it, has been passed to the back of the fauces, the mouth may escape the chemical action of the acid. A medical witness must bear this circumstance in mind, when he is called to examine a young infant suspected to have been poisoned by sulphuric acid. Around the lips and on the neck, may be found spots of a brown colour from the action of the acid on the skin. There is extreme difficulty of breathing, owing to the swelling and excoriation of the fauces and larynx;—and the least motion of the abdominal muscles is attended with increase of pain. These symptoms have been sometimes mistaken for those of disease. (Henke, Zeitschrift der S. A. 1843. ii. 284.) The stomach is so irritable, that whatever is swallowed, is immediately ejected, and the vomiting is often violent and incessant. The matters *first* vomited generally contain the poison: they are acid, and if they fall on a limestone pavement there is effervescence, if on coloured articles of dress, the colour is sometimes altered to a red, or (if logwood) yellow,—the colour is discharged and the texture of the stuff destroyed:—on a black cloth dress, the spots produced by the concentrated acid are brown, and remain moist for a considerable time. An attention to these circumstances may often lead to a suspicion of the real cause of the symptoms, when the facts are concealed. In a case of attempted murder by sulphuric acid in beer, tried at the Lancaster Spring Assizes, 1844, the nature of the poison was suspected from the beer having corroded an apron on which a portion had become accidentally spilled. After a time, there is great exhaustion, accompanied by general weakness:—the pulse becomes quick and small; the skin cold, and covered with a clammy sweat. There is generally great thirst, with obstinate constipation of the bowels;—should any evacuation take place, they are commonly either of a dark brown or leaden colour,—in some instances almost black, arising from the admixture of altered blood. There are sometimes convulsive motions of the muscles, especially of those of the face and lips. The countenance is pale, expressive of great anxiety, and of the most dreadful suffering. The intellectual faculties are quite clear, and death usually takes place very suddenly, in from eighteen to twenty-four hours after the poison has been taken.

THE DILUTED ACID.—When the acid is diluted, the symptoms are much of the same character, but less severe, and not so quickly produced. They vary according to the degree of dilution, the poison acting only as an irritant when much diluted. The vomited matters are not so dark-coloured: in one instance they were nearly colourless. It may be proper here to state, that the diluted sulphuric acid of the London Pharmacopœia contains, in sixteen ounces by measure, one ounce and a half, or one-twelfth part, of concentrated sulphuric acid.

Within what period of time do the symptoms commence?—Most toxicologists, including Orfila (Toxicologie, i. 83, 1843,) Christison (On Poisons, 4th ed. 90,) Galtier, (Traité de Toxicologie, i. 121, 1845,) state that the symptoms commence *immediately*, or during the very act of swallowing, *i. e.* a sense of heat is experienced, with excoriation and burning pain in the throat and stomach. Considering the powerful chemical action of the poison on the thin mucous membrane of the mouth and fauces, it is not easy to understand how there should be any delay in the production of some visible symptoms. In rabbits I have always observed instantaneous effects on the contact of the acid, such as foaming and frothing at the mouth, with a milky-white appearance from the action of the poison on the lining membrane. In all the cases that have hitherto been accurately noticed *from the commencement*, *i. e.* from the act of swallowing, there has been an escape of gas, with severe retching, followed *immediately* or within a few seconds by vomiting. In an instance that occurred to Mr. Tatham of Wandsworth, in which an infant of four months was destroyed by a dose of concentrated oil of vitriol, vomiting of the usual tarry liquid was supposed not to have occurred until *half an hour* or three-quarters of an hour after the poison had been taken, although an alkaline mixture had been given in the meantime. The question relating to the period of occurrence of symptoms, became of some importance at the trial of the case (The *Queen* v. *North*, Guildford Summer Ass. 1846;) for upon the answer to it, rested, in some measure, a charge of murder. Mr. Tatham and myself considered that a teaspoonful of oil of vitriol, whether given by itself or mixed with its bulk of water (in which case the mixture would have a temperature of 196°!,) could not be given by mistake to an infant aged four mouths, without almost immediately producing either vomiting or some symptoms in the infant visible to the person administering it. We considered it impossible that the poison, which must have been taken in a concentrated state, as the contents of the stomach subsequently ejected were quite carbonized, could have remained from a minute to a minute and a half in the mouth and fauces of a young infant, without producing some effects manifest to the most common observer. Then it followed that the defence was unsound, and that no mistake had been made by the mother, as it was alleged, but that the poison had been administered subsequently to the innocent medicine, which the mother swore she had given. An analysis of the medical evidence, as well as an exposure of the sophistry by which the facts of this most important case were perverted, will be found elsewhere. (See post, p. 172, also G. H. Rep., iv. 396, Oct. 1846.) The supposed delay in the occurrence of vomiting in this case rested upon the statement of the mother; but as the medical facts tended to prove that she could not have given the poison, and was not present when it was really administered, it is by no means improbable that a portion had been ejected from the stomach before she saw the child, as this was in the arms of the accused in another room. Without going so far as to say that vomiting must in all cases be immediate, it may suffice to state, that in every well-observed instance yet recorded, it has been one of the earliest symptoms.

From a case observed by Orfila, it appears that even when moderately diluted, there is no delay in the appearance of the symptoms produced by this poison. A man swallowed a certain quantity of sulphuric acid, diluted with its weight (*i. e.* with twice its bulk) of water, and experienced *immediately* the most severe suffering. (Toxicologie, i. 96.) The common opinion of toxicologists, that this poison, from its local chemical action, produces certain effects immediately, is, I believe, correct in all cases in which it is not much diluted with water. It causes some immediate symptoms, and in every case that I have yet met with, speedy vomiting,—a fact borne out by the results of repeated experiments on animals.

The following case strongly supports the view which was taken of this question by Mr. Tatham and myself, on the trial of *North*. On June 6th, at 11 P. M., Mr. Thompson was called in haste to visit an infant of about a year old. The mother had thought it necessary to give her child a little castor-oil for a slight cold. The castor-oil was kept in a closet near some concentrated sulphuric acid, both liquids being contained in the clear bottles commonly used for the oil. A teaspoonful of this oil was first given in a little milk with sugar; but part of the mixture being lost in the administration, it was resolved to give a little more. Half a teaspoonful of sulphuric acid was then given by mistake for castor-oil. This quantity was poured into a teaspoon containing a little sugar and milk, with some remains of the first dose adhering to the spoon, and in this way administered to the infant. *The mistake was immediately discovered by the cries and restlessness of the child;* and by the action of the acid on a coloured dress. The surgeon, who lived two miles distant, saw the child in twenty-five minutes. It was then crying incessantly, with a rough croupy voice. It was restless, agonized with pain, and had occasional vomiting of a tough glairy mucus. Magnesia and milk, with castor oil, were administered freely. The pulse was small and thready. Leeches and a blister were applied to the throat, and mucilaginous demulcents were occasionally given. At 2 A. M. (*i. e.* three hours after the occurrence,) the child seemed to sleep, but there was increased difficulty in the respiration, mucous râle, and rapid pulse. The bowels were freely opened. At 9 A. M. heavy respiration, pulse thready. About 4 P. M. patches of disorganized membrane were coughed up: the child died about 11 o'clock P. M., exactly twenty-four hours after the sulphuric acid had been administered. No post-mortem examination was allowed. (Med. Gaz. xxix. 147.) It will thus be seen that half a tea-spoonful of sulphuric acid was here given to a child a year old, and that certain symptoms *immediately* followed, which led to the discovery of the mistake. In the defence of *North*, it was admitted that a whole tea-spoonful was given to an infant only four months old, and yet it was assumed that no symptoms followed until after the lapse of from one to two minutes!

The local action of sulphuric acid on the *fauces* and *œsophagus* is very energetic: the lining membrane is stripped off in shreds, or peels off in large masses. In a case mentioned by Sobernheim, the lining membrane of the mouth, tongue and fauces, came off in one mass. In another related by Dr. Wilson, the patient, during a violent fit of coughing, brought up a large piece of sloughy membrane, which was found to consist of the inner coat of the œsophagus much thickened and very firm in texture. Its length was eight or nine inches, and its width, that of the œsophagus; it was of a cylindrical form, and pervious throughout its whole extent. (Med. Gaz. xiv. 489.) This has been observed to occur in several other cases. (See p. 170, post.) If the patient survive some days, the motions, which are of a leaden colour, will be found to contain portions of disorganized membrane, from the action of the acid on the stomach and bowels.

The acid produces asphyxia.—This poison may destroy life without reaching the stomach,—a fact sometimes observed in the cases of young children. The larynx is then acted on:—the rima glottidis becomes closed by the swelling of the surrounding parts, and the child dies suffocated. In such cases, death takes place very rapidly. I have found that rabbits, to which this poison was given, died from this cause in the course of a few minutes. Mr. Quain met with the case of a child which became asphyxiated under these circumstances, while he was performing the operation of tracheotomy. The child was recovered by inflating the lungs, but died three days afterwards of bronchitis. On inspection, it was found that the acid had not even reached the œsophagus. (Lancet, Oct. 29, 1836.) Owing to this local action on the larynx, sulphuric acid may easily cause death by suffocation. A similar case is related by Dr. A. T. Thompson, Lancet, June 10, 1837.

On the other hand, Ryland and Porter have remarked that in *suicide* by the sulphuric and other mineral acids, the larynx generally escapes injury. In their view, the epiglottis, during the act of swallowing, completely covers the upper part of the glottis, and thus the acid passes down the œsophagus without affecting the larynx. When the acid has been swallowed by mistake, or when forcibly administered, the larynx is liable to be affected; for so soon as the mistake is discovered, which is almost immediately, all the muscles of the fauces and throat become spasmodically affected, and the fluid is ejected, partly by the mouth and partly by the nares; while, perhaps, a few drops pass at the same time into the glottis, causing inflammation, and rendering tracheotomy necessary. When the poison has been taken voluntarily, the mucous membrane of the mouth, pharynx, œsophagus, and stomach, will present the usual effects of the acid—*i. e.* marks of inflammation and corrosion. When the poison is taken by mistake, the parts chiefly injured are the mouth, pharynx, epiglottis, and sometimes the lips of the glottis; and when forcibly administered to children, there will be symptoms of inflammation of the larynx, accompanied by difficulty of swallowing. Death may take place, as was just now remarked, from this affection of the larynx alone; the acid may not even have reached the œsophagus. (See Ed. M. and S. Journ., xlix. 583; also Med. Chir. Rev. xxviii. 399.) Owing to this local action, cyanosis has been occasionally observed among the symptoms. (Galtier, Toxicologie, i. 192.) Thus, then, as a medico-legal fact of some importance, it is certain that *this poison may destroy life without reaching the stomach.* We cannot, however, say that the discovery of the effects of the poison in the stomach would indicate suicide; because in cases of murder, the stomach has been found disorganized by it, evidently showing that it must have penetrated thus far.

There are at least two instances on record in which this poison has destroyed life in consequence of its having been injected into the rectum by mistake for a clyster. In one case the patient suffered the most acute pain, and died in the course of a few hours. (Med. Gaz. xvii. 623; Annales d'Hyg., 1846, i. 366.)

Can a person who has swallowed sulphuric acid, exert the powers of volition and locomotion?—The severe pain produced by a large dose of this poison, is in many cases sufficient to deprive him of the power of motion. He rolls on the ground in agony. Nevertheless, numerous well-observed facts establish that an individual may sometimes retain astonishing self-command. In the case of *Mr. Schwabe*, who died in twenty-four hours from a dose of six drachms of sulphuric acid, it was proved that the deceased, after having swallowed the acid, beckoned to a cabman, got into a cab, and told him to drive to his house as fast as he could. The deceased had at the time a handkerchief to his mouth, and the only circumstance noticed by the driver was, that he looked very pale. (Med. Gaz. xxxvi. 826.) A case is quoted by Dr. Galtier, in which a man, æt. 52, after having taken some soup, swallowed three ounces of commercial sulphuric acid. He threw himself upon his bed, and it was not until between three and four hours afterwards that the severe pain which he suffered compelled him to seek for assistance. He got up, dressed himself, and was conveyed to the hospital, where he died five hours after swallowing the poison. (Toxicologie, i. 189.) In a more recent instance, a child, æt. 9, swallowed an ounce of oil of vitriol, and although instantly seized with excruciating pains in the throat and stomach, he was able to run home and inform his parents. (Med. Gaz. xxxix. 116.) This retention of power cannot always be referred to the fact of the stomach containing food or liquid sufficient to dilute the poison: because, in Mr. Schwabe's case, the acid was not taken until some time after a meal. The fact is important in a medico-legal view, as the following case will show. In December 1843, a soldier was found lying on the pavement, and suffering from the effects of sulphuric acid. When questioned, he declared that he had been poisoned at a wine-merchant's shop. The man soon died, and the inspection showed that his death had been caused by sulphuric acid, taken probably in a diluted state. None of the acid was discovered in the matter last vomited, or in the stomach of the deceased: that which was *first* vomited had not been collected! Nevertheless, the cause of death was very clear. The wine-merchant's shop where the deceased said he had been poisoned, was at some distance (not specified) from the spot where his body was found; and on the question being put to MM. Ollivier and Chevallier, they gave it, as their opinion, that the deceased could not have exerted a power of locomotion for so great a distance, and affirmed that, in their judgment, based upon cases fatal within a similar period of time, the deceased could have walked only a very short distance after swallowing the poison. They therefore inferred that it was a case of suicide, and not of homicide. (Ann. d'Hyg. 1845, i. 179.) Considering the facts above detailed, and that the sulphuric acid was in this instance diluted, the medical opinion here given appears to have been somewhat stronger than prudence would warrant. (See page 168, post.) An individual who has taken sulphuric acid may undoubtedly retain a power of locomotion; but the degree to which it may be exerted, must depend on the special circumstances of the case.

Can the symptoms produced by this poison cease and re-appear?—In general, it is observed that the symptoms continue to increase in severity until death, when the case is rapid:—but there may be remissions, and, just before death, the pain and suffering have been observed to become considerably abated. With this restriction, then, it appears to me the question should be answered in the negative. The following case, mentioned by Dr. Johnson, at a meeting of the Westminister Medical Society, in October 1836, is in this respect curious. A person swallowed two ounces of concentrated sulphuric acid. After suffering from severe symptoms, the patient rallied and apparently recovered. A few days afterwards, during a severe fit of coughing, he brought up a quantity of the acid in its pure state:—the acid having been a fortnight in the stomach! It produced, in its passage upwards, fatal inflammation of the larynx. On inspection, it was ascertained that the acid had been surrounded by a cyst, formed by secretions from the stomach, which burst during the fit of coughing. This is a most extraordinary case, and, so far as I know, unexampled in the history of poisoning. In explanation it has been suggested, that when concentrated sulphuric acid is dropped guttatim into albumen, a cyst of coagulum forms around the globules of acid, and preserves the remainder from its action. This effect, however, is only temporary, and it will not satisfactorily account for a large quantity of the poison being swallowed and retained in the stomach for a fortnight.

Among the *secondary* symptoms of poisoning by this acid, when the individual survives some days or weeks, should be mentioned profuse salivation. This was observed in Mr. Tatham's case. (G. H. Rep. iv. 396, Oct. 1846.) Salivation commonly occurs about the second or third day—sometimes later. Desgranges observed a miliary eruption on the skin among the secondary consequences of poisoning by sulphuric and nitric acids. (Belloc, Cours de Méd. Lég. 120; Galtier, Traité de Toxicologie, i. 176.)

POST-MORTEM APPEARANCES.

It has been already remarked, that these are not always to be found in the stomach; they may be confined to the region of the fauces and larynx. In an inpection of the body, the whole course of the alimentary canal, from the mouth downwards, ought to be examined; since in all recent or acute cases, it is in the œsophagus and fauces that we obtain strong evidence of the action of a corrosive poison. The discovery of the usual marks of corrosion in these parts, is always highly corroborative of the signs of poisoning found in the stomach. During the inspection, the examiner must not omit to notice any spots on the skin produced by the action of the acid;—these are commonly of a dark brown colour, and are situated about the mouth, lips, and neck. The appearances met with in the body will vary according to whether death has taken place rapidly or slowly. Supposing the case to have proved fatal very rapidly, the membrane lining the *mouth* will be found white, softened, and corroded; but this appearance may be absent. It was just now observed, that when the poison has been administered by a spoon, the mouth may escape the chemical action of the acid. In the case of the *Queen* v. *Thomas* (Monmouth Lent Assizes, 1847,) it was proved that the fauces, œsophagus, and stomach of the deceased, an infant ten days old, were much corroded by sulphuric acid, which had been given to it in a somewhat diluted state; but there was no appearance of injury to the mouth. This was probably owing to a spoon having been used, and the poison having been poured down the throat slowly, as the mucous membrane was extensively corroded at the back part; and it was clear, therefore, that some corrosive substance had passed into the fauces. The mucous membrane of the *fauces* and *œsophagus* will commonly be found corroded, having sometimes a brownish or ash-grey colour. The corroded membrane of the œsophagus is occasionally disposed in longitudinal plicæ, portions of it being partly detached. The *stomach*, if not perforated, is collapsed and contracted. On laying it open, the contents are commonly found of a dark brown or black colour, and of a tarry consistency, being formed in great part of mucus and altered blood. The contents may or may not be acid, according to the time the patient has survived, and the treatment which has been adopted. On removing them, the stomach may be seen traversed by black striæ, or the whole of the mucous membrane may be corrugated, and of a dark brown or black colour. This blackness is not removed by washing. On stretching the stomach, traces of inflammation may be found between the rugæ, indicated by a deep crimson-red colour. On removing the blackened membrane, the red colour indicative of inflammation, may be also seen in the parts beneath. Both the dark colour and marks of inflammation are sometimes partial, being confined to insulated portions of the mucous membrane. When the stomach is perforated, the coats are softened, and the edge of the aperture is commonly black and irregular. In removing the stomach, the aperture is apt to be made larger by the mere weight of the organ. The contents do not always escape; but when this happens the surrounding viscera are attacked by the poison. In a case which occurred at Guy's Hospital, the spleen, the liver, and the coats of the aorta, were found blackened and corroded by the acid, which had escaped through the perforation. Dr. Craigie of Edinburgh thinks that even when there is no perforation of the stomach, the acid may find its way by transudation through the coats of the organ, in a very short time after it has been swallowed. In a case in which two ounces of the strong acid had been swallowed, and the person died in three hours and a half, he found that the peritoneum and the fluid contained in it, reddened litmus paper strongly. There was also a slightly acid reaction even in the serous membranes of the thorax. It does not appear, however, that the nature of this acid was determined by the application of any test. The *small intestines*, in acute cases, are found more or less inflamed; and their contents are of the same nature as those met with in the stomach.

It is important for the medical witness to bear in mind, that the condition of the fauces and œsophagus above described, is not constantly met with. Strange as it may appear, cases are recorded in which, notwithstanding the introduction of the poison into the stomach, the œsophagus has escaped its chemical action. Reliance might be wrongly placed upon this absence of corrosion as positive evidence that sulphuric acid could not have been swallowed; and, therefore, in this respect, a case reported by M. Blondlot of Nancy is of especial interest. This gentleman was required to examine the clothes and viscera of an infant named *Boullet*, aged two months, that had died from the effects of sulphuric acid. The tongue, pharynx, and œsophagus presented *no mark of corrosion*, or any pathological appearance indicating that a corrosive substance had been in contact with them. There was no eschar or alteration of colour in any part. The appearances in the stomach were not very striking. A careful chemical analysis showed that sulphuric acid existed abundantly on the clothing, but not a trace of the poison could be detected in the viscera. The case was remitted to MM. Devergie, Barse, and Lesueur for examination: they confirmed the conclusions of M. Blondlot, and pronounced an opinion that, notwithstanding the absence of marks of corrosion in the viscera, and of the acid from their contents, the deceased had died from sulphuric acid administered to it. They were inclined to attribute the absence of the poison to vomiting and elimination by the urine. (Journal de Chimie Medicale, 1846, ii. 17. See also ante, p. 31.)

This case occurred in April 1845; and it may be well to contrast it with that of *Thomas*, tried at the Monmouth Lent Assizes, 1847. The deceased infant in this instance was ten days old. The mother was charged with having administered sulphuric acid to it, and caused its death. The œsophagus, stomach, and intestines were more or less corroded, but the *mouth* had escaped the action of the acid. No sulphuric acid was found in the viscera or their contents, but it was abundantly detected on the clothes of the child. The two cases are therefore very similar, and only differ in the fact, that there was much more decided evidence of corrosion by sulphuric acid, in the English than in the French case! The counsel who defended the case of *Thomas*, contended that "no 'traces of unfairness' were to be discovered in the mouth; and how could a poison so strong and disagreeable to the taste, be got down the throat of an infant so young without leaving some ill effect behind in the mouth of the child." The judge who tried the case is reported to have told the jury, that the evidence of medical witnesses was "generally a matter of conjecture or guess," and that the "traces of unfairness" (chemical corrosion) in the œsophagus and stomach "might have been produced by other substances (!) and not by the poison itself! Hence the reader will not be surprised to learn, that in the case in which the evidence of death from sulphuric acid was strongest, the accused party was triumphantly acquitted; while, in that in which it was less cogent (*i. e.* in the French case,) but better sifted and appreciated, the prisoner was convicted and condemned to hard labour for life. These conflicting results cannot be right. Either *Thomas* was acquitted upon an entirely mistaken view of medical facts, or the prisoner *Boullet* was most improperly convicted!

When the poison has been taken in a *diluted* state, the marks of inflammation on the mucous membrane are more decided, and the charring is not so considerable. Nevertheless, the acid, unless too much diluted, acts upon and darkens the blood in the vessels, as well as that contained in the stomach, although it may exert no carbonizing action on the mucous membrane, or on the contents.

Chronic Poisoning.—The appearances just described will not, of course, be met with in protracted cases. If the individual survive sufficiently long, all signs of inflammation and corrosion will disappear. Thus, in the interesting case reported by Mr. Tatham, in which the child survived twenty-five days, the mucous membrane of the mouth and fauces was sound but pale: that of the œsophagus, stomach, and duodenum was smooth, and equally free from any marks of corrosion or inflammation. (G. H. Rep., Oct. 1846, 396.) In other instances the mucous membrane has been found entirely destroyed, or more or less ulcerated. This destruction of the inner coat of the stomach leads to death, by impairing the function of digestion. In several cases, the aperture of the pylorus has been found much contracted. Sometimes there will be stricture of the œsophagus. The common secondary causes of death in these chronic cases, are fever, irritation, or exhaustion of the system.

Absorption.—It has been a disputed question, whether sulphuric acid is or is not absorbed and carried into the circulation in cases of acute poisoning. M. Bouchardat considers that it is absorbed, and that it causes death by leading to a coagulation of the blood in the heart, aorta, and large blood-vessels. He has found these coagula in two cases in considerable quantity; and in one of them, the lining membrane of the aorta was reddened. (Annales d'Hygiène, 1837, i. 362.) I have observed this last-mentioned appearance in one case, as well as the occurrence of coagula in two instances; but there does not seem to be any reason for believing that they result from the action of a portion of sulphuric acid absorbed. In analysing these coagula taken from persons who had been killed by sulphuric acid, I have never found a trace of that acid present in them. According to Orfila, the absorption of these mineral acids may take place owing to their compounds with albumen being soluble. There is no doubt that all these albuminous compounds are soluble in a large quantity of water, but they are insoluble when much acid is present. In a case reported by Dr. Letheby to the Pathological Society, a chemical analysis of the urine proved that the acid was rapidly eliminated by this secretion. The quantity thus passed within four days was considerable. (Med. Gaz. xxxix. 116; ante, p. 31.)

QUANTITY REQUIRED TO DESTROY LIFE.

The dangerous effects of this poison appear to arise more from its degree of concentration, than from the absolute quantity taken. The quantity actually required to prove fatal, must depend on many circumstances. If the stomach be full when it is swallowed, the action of the acid may be spent on the food and not on the stomach; and a larger quantity might thus be taken, than would suffice to destroy life if the organ were empty. In one case, one drachm of sulphuric acid destroyed life in seven days:—in another (*Humphrey's* case, Med. Gaz. viii. 77,) about one drachm and a half destroyed life in two days. In *Mr. Schwabe's* case, six drachms destroyed life in twenty-four hours. (Med. Gaz. xxxvi. 826.) In one instance, a patient survived fifty-five hours after taking three fluid ounces of the concentrated acid (Dr. Sinclair, Med. Gaz. viii. 624:) in another related by Sobernheim, a man swallowed an ounce and a half of the concentrated acid, and yet slowly recovered from its effects. (Handbuch der Prakt. Tox. 384.) In a case quoted by Dr. Craigie, a young woman aged eighteen, recovered after having taken *two ounces* of concentrated sulphuric acid. She was completely restored in about eighteen days. (Ed. Med. and Surg. Jour., April 1840.) Another instance of recovery after two ounces of the concentrated acid had been taken, is reported by Mr. Orr. (Med. Gaz. iii. 255.) A remarkable instance of recovery from a large dose was observed in a case which occurred in the practice of M. Biett. The patient, a man aged 31, swallowed by mistake *three ounces* (by weight?) of commercial sulphuric acid. Severe burning pain and vomiting immediately followed; the man fell and rolled on the ground in agony, but nevertheless was able *to walk some distance* to the hospital without assistance, although he rested occasionally. Milk and magnesia were freely given to him, and in a week he perfectly recovered. The most striking symptom was excessive salivation, which set in on the second day, and continued for three days. (Galtier, Toxicologie, i. 186.) It is probable that, in these instances of recovery from large doses, the greater part of the poison is expelled in the matter first vomited. In Dr. Letheby's case, the patient, a child only nine years old, recovered in a short time, after having swallowed one ounce of concentrated sulphuric acid. In this case nothing was done for five minutes: for the first few days the patient was copiously salivated. (Med. Gaz. xxxix. 116.) The smallest quantity which I have been able to meet with as having proved fatal, was in a case already quoted. Half a tea-spoonful of concentrated sulphuric acid was given to a child, about a year old, by mistake for castor-oil. The usual symptoms came on, with great disturbance of the respiratory functions; and the child died in twenty-four hours. The quantity here taken could not have exceeded *forty drops.* (Med. Gaz. xxix. 147; see also ante, p. 162.) It is however, doubtful whether this small quantity would have proved fatal to an adult. The smallest fatal dose which Dr. Christison states he has found recorded, was *one drachm;* it was taken by mistake, by a stout young man, and killed him in seven days. (Op. cit. 162.)

PERIOD AT WHICH DEATH TAKES PLACE.

It has been already stated, that the average period at which death takes place in cases of acute poisoning by sulphuric acid, is from eighteen to twenty-four hours. When the stomach is perforated by it, it proves more speedily fatal. In one instance, reported by Dr. Sinclair, a child about four years old died in four hours—the stomach was perforated. When the poison acts upon the larynx, death may be a still more speedy consequence from suffocation; and owing to this, it appears to be more rapidly fatal to children than adults. Dr. Craigie mentions a case in which three ounces of concentrated sulphuric acid destroyed life in three hours and a-half; but the shortest case on record is, perhaps, that mentioned by Remer in Hufeland's Journal. In this instance death took place *in two hours.* A case is reported by Mr. Watson, in which a woman swallowed two ounces of the strong acid. She died in *half an hour,* but it appears that a quarter of an hour before death she had made a deep wound in her throat, which gave rise to great hæmorrhage. The stomach was found very extensively perforated:—but it is highly probable that the wound accelerated death in this case.

On the other hand, there are numerous instances reported, in which the poison proved fatal from secondary causes, at periods varying from one week to several months. In Mr. Tatham's case, the child recovered under very judicious treatment from the first effects, but died of starvation after twenty-five days, from the impossibility of retaining any kind of food on its stomach. (G. H. Rep., Oct. 1846, 396.) A very remarkable instance of this kind occurred to Dr. Wilson of the Middlesex Hospital, and is referred to by Mayo in his Outlines of Pathology. A young woman swallowed about a table-spoonful of sulphuric acid on the 4th of January, and died from its effects on the œsophagus, on the 14th of November following. She gradually wasted away, and died from innutrition. This was forty-five weeks, or *eleven months*, after she had swallowed the poison. There is no doubt that the acid may prove fatal at all intermediate periods, and at intervals much longer than this; but the longer this event is protracted, the more difficult will it become to ascribe death to its effects.

TREATMENT.

Calcined magnesia or the carbonate of magnesia, finely levigated and mixed with milk or water, may be exhibited as speedily as possible. In the absence of these remedies, finely powdered chalk or whiting may be given. Although it is the general practice to recommend magnesia and chalk, it appears to me, from a case which I lately had the opportunity of examining, that a solution of carbonate of soda or potash, properly diluted, would act more effectually and more speedily in neutralizing the poison. The insoluble particles of magnesia adhere closely to the mucous membrane, and do not readily come into contact with the acid. In examining the dark tarry matter vomited by a child half an hour after the concentrated acid had been taken, I found it still intensely acid, although during the whole period, a magnesia mixture had been freely given in divided doses. This objection would not apply to the use of bicarbonate of magnesia or lime; and the evolution of carbonic acid would be a minor evil compared with the action of sulphuric acid in an unneutralized or imperfectly neutralized condition. Sobernheim and Simon relate several instances in which persons who had taken this poison, were apparently saved by the free use of these alkaline diluents. In the absence of these substances, oil may be freely administered. Carbonate of magnesia has been sometimes beneficially given mixed with oil. There is often very great difficulty in making the patient swallow:—the throat being swollen, and blocked up with shreds of tough coagulated mucus and sputa. Hence it has been recommended to employ the stomach-pump for the purpose of injecting the liquids into the stomach. The use of this instrument ought, however, if possible, to be avoided; since it is only likely to lacerate and perforate the structures which are softened and corroded by the acid. When there are symptoms of suffocation from an affection of the larynx, tracheotomy must be immediately resorted to. On the whole, the antidotal treatment of cases of poisoning by sulphuric acid has not been very successful, the patient not having been seen sufficiently early by a medical man to give much hope of success. It should be remembered, that the poison begins to act instantly on contact; and if the stomach be at the time empty, there is but little prospect of saving the patient. We often find these cases proving fatal even when every trace of the poison has been removed from the stomach, owing to the extensive changes produced, and the sympathy with remote organs.

That this antidotal treatment may, however, occasionally be the means of saving life, the following case, related by Barzellotti, will show. A man aged 40, swallowed by mistake a quantity of the oil of vitriol, and was brought to the hospital of Santa Maria Nuova. He was suffering from intense burning pain in the throat and abdomen, as well as from other severe symptoms; calcined magnesia in water was given to him at short intervals, until it was supposed enough had been taken to neutralize the acid. A quantity of tepid water was then administered to promote vomiting; and, on examining the vomited matters, it was found that the sulphuric acid was neutralized by magnesia. The patient was then bled; leeches and fomentations were applied to the epigastrium, and demulcents exhibited. The man slowly recovered, suffering from difficulty of swallowing and severe cough. In one fit of coughing he expelled a mass of false membrane, of the form and size of the œsophagus. The abdomen and throat were tender at the time of his discharge. (Questioni di Medicina Legale, ii. 307.) It is to be observed, that cases of accidental poisoning like this, much more frequently do well than those of suicide—the quantity of poison swallowed being in general small.

A case lately reported by Dr. Borgstedt of Minden, shows the best mode of after-treatment. A boy, aged three years, swallowed from one to two drachms of oil of vitriol. Some train-oil was given to him. When medical assistance was procured, the patient was lying speechless and motionless on his back, face pale, eyes deeply sunk and closed, breathing difficult, and accompanied by a rattle. The skin around the mouth, as well as the lining membrane, had been destroyed by the acid. Carbonate of potash was given at intervals, and this was followed by the vomiting of a dark-brown slimy matter. When vomiting had ceased, oily emulsion, with carbonate of magnesia, was exhibited every half-hour. Strong febrile symptoms set in, with severe pain in the region of the stomach. Leeches were applied, and repeated for three days. The eschars from the destroyed skin and membrane were smeared with olive oil and yolk of egg, with great relief to the patient. The only nourishment allowed was milk. The fever and abdominal tenderness disappeared about the fourteenth day. On the first day the motions had a natural colour. From the second to the seventh day they were very hard, and appeared like slaked lime; from the seventh to the thirteenth day they assumed an ash-grey colour; and on the fourteenth day they had their usual characters. (Casper's Wochenschrift, May 9, 1846.)

The following case of successful treatment, which was reported by Mr. Gardner to the Lancet, Aug. 25, 1838, deserves to be here mentioned. A young man swallowed half an ounce of strong sulphuric acid. The usual symptoms appeared; milk and carbonate of magnesia were freely given. This person recovered in twelve days. One of the secondary symptoms was profuse salivation.

It is worthy of remark, that several cases of recovery have taken place, where no chemical antidotes were administered. The treatment consisted simply in the exhibition of large quantities of gruel and milk; and there is no doubt, that any thick viscid liquid of this description, as, for example, linseed oil, or flour and water, must be beneficial, by combining with the acid and arresting its corrosive effects. In short, such a liquid would act much in the same way as the presence of a large quantity of food is known to act, when the acid is swallowed soon after a meal. In all cases, it would be advisable to combine the use of chemical antidotes, with the copious administration of mucilaginous drinks.

CHEMICAL ANALYSIS.

This acid may be met with either concentrated or diluted; and a medical jurist may have to examine it under three conditions:—1. In its simple state. 2. When mixed with organic matters, as with liquid articles of food or in the contents of the stomach. 3. On solid organic substances, as where the acid has been thrown or spilled on articles of dress or clothing.

In the simple state.—If *concentrated*, it possesses these properties:—1. A piece of wood or other organic matter plunged into it, is immediately carbonized or charred. 2. When boiled with wood, copper-cuttings, or mercury, it evolves fumes of sulphurous acid; this is immediately known by the odour, as well as by the acid vapour first rendering blue, and then bleaching starch-paper dipped in a solution of iodic acid. 3. When mixed with an equal bulk of water, great heat is evolved (nearly 200° F. in a cold vessel.)

Carbonization of Organic Matter by Sulphuric Acid.—Concentrated sulphuric acid, it is well known, possesses the property of abstracting the elements of water from most organic substances, and thus setting free carbon, whereby it becomes darkened. This property is more remarkably manifested with respect to sugar than any other substance. Mr. Phillips states that 1-100th of a grain of sugar is sufficient to discolour a fluid-ounce of sulphuric acid, even without the aid of heat; and if the sugar be in moderately large quantity; the mixture, on a little water being added to dissolve it, is perfectly blackened. This property of carbonizing organic matter is lost, when the acid is diluted even in a moderate degree; and when it is not manifested with respect to sugar, it is, for the reason above stated, not likely to take place when the acid is mixed with any other organic substance. Thus, sugar and a piece of deal-stick (woody fibre) are both blackened by the concentrated acid, but the sugar only is blackened when the strength of the acid is lowered by the addition of water. As this power of sulphuric acid to carbonize sugar became a very material question on a recent trial for poisoning by oil of vitriol, I subjoin the account of some experiments on the subject.

1. On putting a lump of sugar into a drachm of concentrated sulphuric acid in a white cup, it speedily acquired a yellowish colour wherever it was wetted by the acid. On adding *one drachm* of water, and mixing,—the whole of the liquid became black, frothy, evolved vapour, and, a thermometer plunged into it, indicated a temperature of 180°. A common piece of deal-stick was not carbonized in this mixture, but it had merely a greenish-yellow colour when washed from the loosely adhering carbon of the sugar. If the same proportions of acid and water be *mixed* together *before* the addition of the sugar, a piece of deal-stick is scarcely affected; and when sugar is added, it is only after two or three minutes that the mixture acquires at first a yellowish, and then a reddish-brown colour, but no carbon is set free. The mixture had a temperature of 196°.

2. In this case *two drachms* of water were added to the acid in which the lump of sugar had been placed. The sugar became first yellow, then black, and, on gently shaking the vessel, the whole of the mixture became black, and acquired a temperature, in one experiment, of 164°, and in another of 160.° A piece of stick plunged into this mixture was neither corroded nor carbonized. The same proportions of acid and water were previously mixed, and sugar then added. There was scarcely any perceptible change of colour for several minutes, but the sugar dissolved in the mixture, (diluted sulphuric acid) which had a temperature of 160°. In the course of an hour it had acquired a reddish-brown colour.

3. One drachm of sulphuric acid was placed in a white cup, and two drachms

of water were added. A small lump of sugar was then dropped into the mixture. In a few seconds, the sugar became blackened; and on shaking the mixture, the whole formed a deep black liquid, at a temperature of 160°.

4. Two drachms of water were placed in a cup, and one drachm of sulphuric acid added. The acid fell through the water, and, on putting into the liquid a small lump of sugar, this was darkened in a few seconds, and gave, when shaken, a greenish-black colour to the whole of the liquid, which had a temperature of 160°. The result of this experiment will of course depend upon the degree with which the acid mixes with the water in falling through it.

Thus, then, it will be seen, that even by putting acid and water, or water and acid, before the sugar, the carbonizing action of the acid is well marked. It is only when the acid and water are *thoroughly intermixed before* the addition of the sugar, that the liquid does not become blackened.

5. When *three drachms* of water were added to one drachm of sulphuric acid, in which a lump of sugar was immersed, the liquid immediately darkened around the sugar, and the whole speedily acquired a blackish-green colour. The temperature was 156°.

6. When *four drachms* of water were added to one drachm of oil of vitriol, in which a lump of sugar was immersed, black streaks speedily began to appear around the sugar. On slight agitation, the whole mixture acquired a greenish-black colour, and the temperature was 142°. In the two last cases, a stick plunged into the mixtures was neither corroded nor carbonized in any degree.

Thus, then, with two, three, and even four parts, by measure, of water, there are well-marked effects, both in change of temperature and colour.

The case in which this question respecting the action of sulphuric acid on sugar arose, was that of *Mary North* (Guilford Summer Ass. 1846,) tried for the murder of an infant by giving to it oil of vitriol. The deceased had died from the effects of the poison; therefore the only part of the case which created difficulty, was the proof of administration. The mother of the deceased, wishing to give the child some aniseed-spirit and water, placed a lump of sugar in a white cup, and added a tea-spoonful of the spirit; she then went to another apartment, and poured from a kettle about a tea-spoonful, but certainly not more than two tea-spoonfuls, of water. She observed no particular appearance in the mixture; she tasted it, and there was no hot or acid taste; she then gave about two tea-spoonfuls of it to the infant, while a little girl who was present drank up the dregs, and suffered no ill effects. The prisoner was present, and in about half a minute took the child. After the child had taken the liquid, there were no symptoms or effects to attract attention, and the child appeared relieved of the wind from which it had suffered. The mother left the room, and the prisoner took the infant into an adjoining pantry, in which it was sworn there was a bottle of vitriol, put there by the prisoner. In about a minute and a half or two minutes, the mother, owing to a noise, returned to the room, and found the infant evidently writhing in great pain, its mouth covered with a whitish froth. The prisoner, according to one witness, while bringing it from the pantry into the kitchen, was in the act of wiping the child's mouth. Medical assistance was immediately sent for; but in spite of the best treatment, the child died. These facts were sworn to in evidence; and the mother, the prisoner, and the girl (who drank the remainder of the liquid in the cup,) told Mr. Tatham, a medical witness, on the evening of the occurrence, that aniseed only had been given to the child; and all agreed that the aniseed and sugar were mixed together in a cup, and the water afterwards added. As neither the prisoner nor the mother could possibly know the difference in the chemical results, according to the order in which these substances are mixed, and the prisoner could not, only an hour after the occurrence, have had any suggested motive for stating that the water and aniseed were mixed first, we are bound to take this as a true statement of the facts.

The defence was, that the mother had made a mistake, and given a tea-spoonful of oil of vitriol in place of aniseed. Mr. Tatham, the medical witness, when questioned respecting the effects produced by mixing vitriol and water, very properly, in obedience to his oath of swearing to the *whole* truth, called the attention of the learned judge to the fact, that had the mother used oil of vitriol in place of aniseed, in the order in which the articles were sworn to have been put together, the liquid would have acquired a very high temperature, and have become blackened, so that she could not have administered it to her infant without being made fully aware of the mistake. [This statement is perfectly borne out by the results of the experiments already mentioned.] The counsel for the prisoner, however, requested the witness to perform an experiment in Court, by mixing together *the acid and water*, putting *double* the quantity of water, and to add the sugar *last*. The result of course was, that the mixture became only slightly darker, but was not blackened, for the acid was already *diluted*. This experiment of the learned counsel's completely deceived the jury, for it was concealed from the Court, that the order of mixing suggested, was exactly the reverse of that sworn to in evidence by unscientific witnesses. Hence it was argued that the mother had made a mistake, and that, in order to conceal this, she had charged the prisoner with the crime of murder!

The perversion of chemical evidence in this case was not a little remarkable; the irrelevant results doubtless had its influence with the jury, and they were led away with the false impression, that whether oil of vitriol, or a colourless aniseed mixture, had been used in this instance, there would have been no difference in the appearance of the liquid on mixture! The fact that the contents of the stomach were completely *charred* to a tarry liquid renders it unnecessary to discuss the question, whether the acid swallowed by the child would, or would not, carbonize sugar; since, according to my experiments, which are only confirmatory of those of other chemists, sulphuric acid, which is so diluted as not to carbonize sugar, will certainly not char blood, mucus, or mucous membrane.

The attorney for the prisoner, relying upon his knowledge of chemistry, subsequently published a statement in which he argued that the innocence or guilt—indeed the hanging of the accused—depended on the simple fact, whether *sugar would, or would not, be carbonized under the circumstances!* If this view be correct, and if there be any faith in chemical facts, he has unintentionally, but most conclusively, proved that the accused was guilty, and that she was acquitted upon a mistake: for when the ingredients are put together in the order and proportions sworn to, the mixture becomes invariably blackened, whether the acid, sugar, or water, be added first; always provided the acid and the water be not in the first instance well shaken and mixed. If, however, an individual be permitted to assume that the three substances were mixed in an order different to that repeatedly sworn to, and to draw an inference from this assumption, it would be as well to assume at once that no sugar was used. It appears to me, that we are bound to take the experiment according to the evidence, when, from a want of chemical knowledge, there cannot be the slightest suspicion of a motive for fabrication,—or to reject it altogether. We have no right to twist facts of this kind to suit our own particular views. The result of this investigation appears to me to show that a Court of Law should exercise the greatest caution in not allowing itself to be imposed upon by chemical experiments entirely at variance with the facts stated in evidence. Let us suppose that it had been sworn by the witnesses that the liquid (aniseed or vitriol) had been well mixed with water *before* the sugar was added,—Would a medical witness have been permitted, either by the attorney or counsel for the accused, to express an opinion unfavourable to the prisoner from results obtained by an *inverse mode* of mixing the ingredients? Assuredly not. Yet, what would have been denounced as a gross deception in *medical* evidence, was received without comment as *legal* evidence in favour of the accused!

It is most probable, that the jury, in acquitting the prisoner, disbelieved the evidence; for if the facts were true, to suppose that the mother made a mistake, and then imputed the foul crime of murder to an innocent person, involves the following assumptions, which are, in my judgment, contrary to medical experience. 1. That sulphuric acid, mixed as stated in evidence, does not carbonize sugar. 2. That two teaspoonfuls of such a mixture, at a temperature of nearly 200°, may be poured into the mouth of an infant of four months without producing, within a minute, any symptom or effect to indicate a mistake! 3. That symptoms with a whitening of the thin membrane of the mouth would only appear after the lapse of from one to two minutes. 4. That although the acid administered, would not (on the hypothesis of the defence) carbonize sugar, it would be strong enough to carbonize the blood, mucus, and mucous membrane (ejected from the stomach) and to produce extensive eschars in the mouth and fauces, as well as on the skin externally!

A very full report of this trial will be found in the Guy's Hospital Reports, Vol. iv. p. 396. It is well calculated to show how medical facts of the greatest importance may be misunderstood and misrepresented in a Criminal Court. A grave attack was made upon Mr. Tatham's evidence in the Lancet (Sept. 12, 1846) for his conscientious performance of a painful duty on this occasion. Resting upon an *ex parte* statement by the attorney for the prisoner, a writer in this journal says—"he (the attorney) is entitled to great credit for the "tact and ability" which he displayed in this case. The life of the innocent accused person was saved by the sagacity of the attorney." This kind of *tact and ability*, when exerted in perverting the medical facts of a criminal case, and in placing the results of a false and irrelevant experiment before a jury as if it were a true representation of the facts, may, however, operate in two ways: it may not only lead to the acquittal of a guilty person—but to the conviction and execution of one who is innocent! The less tact and ability, thus displayed, and the more truth and honesty we have in reference to medical evidence on charges of murder, the greater will be the security that the detestable crime of secret poisoning which has of late years spread throughout this country to a most lamentable extent, does not go unpunished.

The Diluted Acid.—For the acid in the *diluted* state, but one test need be applied:—a solution of a salt of barytes,—the *Nitrate of barytes*, or the *Chloride of barium*. Having ascertained by test paper, that the liquid is acid, we add to a portion of it, a few drops of nitric acid, and then a solution of nitrate of barytes. If sulphuric acid be present, a dense white precipitate of sulphate of barytes will fall down—which is insoluble in all acids and alkalies. If this precipitate be collected, dried and heated to redness in a small platina crucible with five or six parts of charcoal powder, it will, if a sulphate, be converted to sulphuret of barium. To prove this, we add to the calcined residue, diluted muriatic acid, at the same time suspending over it, a slip of filtering paper moistened with a solution of acetate of lead, or, what is exceedingly convenient, we place the residue on a slip of glazed card (coated with carbonate of lead,) scraped and wetted on the surface. (The card should be first tested for lead; because some kinds of glazed cards are made without lead.) If the original precipitate were a sulphate, the vapour now evolved will be sulphuretted hydrogen, known by its odour, and by its turning the salt of lead or staining the card of a brown colour. Instead of charcoal, we may use an equal bulk of cyanide of potassium as the reducing agent, and the experiment may then be performed in a small reduction tube over a spirit lamp. On breaking the tube and placing the powder on a glazed card (containing lead) previously wetted, the stain of sulphuret of lead will be perceived;—or the calcined residue may be dissolved in water and tested. The smallest visible quantity of sulphate of barytes thus admits of easy detection.

The delicate action of this test is such, that a solution containing not more than the 1-25,000th part by weight of sulphuric acid, is precipitated by it. When the sulphuric acid is diffused through a minimum of water, the barytic test gives a perceptible precipitate with the 1-110th part of a grain of the acid. If, however, this small quantity be diluted with an ounce of water, the test produces no perceptible change. In these experiments, distilled water must be used, since all kinds of river and spring water are precipitated by the test. With regard to the reduction of the precipitate to the state of sulphuret by charcoal or cyanide of potassium, I have found that one-half grain of the sulphate of barytes will yield satisfactory evidence; and a quarter of a grain will give traces of sulphur, although somewhat indistinct. This is equivalent to about one-eighth of a grain of common oil of vitriol (bihydrate.) In cases of poisoning, however, we either find the acid in much larger proportion, or it is altogether absent. Orfila recommends that the diluted acid should be concentrated by evaporation and then treated with metallic copper, to liberate sulphurous acid; but this process is more troublesome and less likely to prove satisfactory than that just described.

Objections to the tests.—When any inference is drawn by a medical witness from the presence of a minute quantity of this acid in a suspected liquid, it might be fairly objected that some portion of sulphuric acid had become accidentally introduced during the experiment. Thus the nitric acid used, may have been contaminated with sulphuric acid;. or the wood-charcoal may itself have contained some saline sulphates, which would lead to the production of an alkaline sulphuret. The purity of these substances should then be determined by separate experiments. Again, too much nitric acid must not be added to the liquid before applying the test:—because the salt of barytes is insoluble in strong nitric acid, and a white precipitate therefore falls (crystalline nitrate of barytes,) although no sulphuric acid be present. The obvious remedy for this, is to dilute the liquid with water before performing the experiment; or if a doubt exist, afterwards—when any precipitated sulphate of barytes will be left, while any portion of precipitated nitrate will be redissolved.

But the question arises—Are there no other liquids liable to be precipitated by this test, and lead thereby to a fallacious inference? Nitrate of barytes is precipitated by other acids—namely, the sulphurous, fluosilicic, selenic, and iodic. The last is not precipitated by the test if it be diluted, and the nitric acid be first added: therefore it can constitute no objection to the process here recommended. The three first form precipitates insoluble in nitric acid; but the fluosilicic acid only when moderately concentrated, and then more slowly than the sulphuric acid. If the fluosilicic acid be much diluted with water, and nitric acid first added, the test gives no precipitate with it. All objection on these grounds is, however, removed by the fact, that sulphurous acid is immediately recognised by its odour of burning sulphur, and may be separated from any sulphuric acid mixed with it by simply boiling it:—and with respect to the fluosilicic and selenic acids, the white precipitates formed by them, calcined with charcoal or cyanide of potassium, and digested with an acid, do not evolve sulphuretted hydrogen, or act on a salt of lead like that formed by sulphuric acid. Besides, it is not probable that such substances as the fluosilicic, selenic, and iodic acids, should ever be met with in common life, or find their way out of a chemical laboratory. The iodate, fluosilicate, and seleniate of barytes do not, like the sulphate, yield a sulphuret when calcined with charcoal.

Acid saline solutions. But there are other objections:—1. A solution of alum, or of any acid sulphate, might be erroneously pronounced to be free sulphuric acid; for alum would give all the re-actions with the tests which have been here described. The answer to this objection is very simple; we must slowly evaporate a portion of the suspected liquid in a watch glass—there will be a saline residue if it be a solution of alum, otherwise not: for sulphuric acid should be entirely dissipated by heat, or it should leave only the faintest traces of sulphate of lead. 2. The quantity of free sulphuric acid present might be erroneously estimated, in consequence of some simple medicinal sulphate (as Epsom salt) being mixed with it. This may be determined also by evaporation; and the free sulphuric acid separated by warming the liquid, and adding finely-powdered carbonate of barytes, until effervescence ceases. The precipitate formed would be sulphate of barytes, and represent the free sulphuric acid present.

There is, however, another source of error: any acid mixed with a common sulphate employed in medicine might be mistaken for free sulphuric acid; as, for example, a mixture of citric or acetic acid with sulphate of magnesia. This may always be suspected when any saline residue is left on evaporating the mixture. In such a case carbonate of barytes would not separate the free acid, for it would form a soluble barytic salt with the extraneous acid; and this, by re-acting on the sulphate of magnesia, would precipitate the sulphuric acid of that salt, and thus lead to error. Several methods have been proposed to remove this difficulty: the following is perhaps the best. Procure by evaporation and calcination the whole of the saline sulphate from a measured quantity of the liquid. Re-dissolve this in water, acidulate the solution with nitric acid:—precipitate all the sulphuric acid of the salt by nitrate of barytes, then dry and weigh the sulphate thus procured. Next obtain from an equal quantity of the liquid before evaporation, the whole of the precipitate produced on adding to it the nitrate of barytes and nitric acid—dry it, weigh it, and compare its weight with that derived from the sulphate of the evaporated liquid. It is obvious, that if there be no free sulphuric acid present, the weights will be the same in the two cases:—but should there be any, its quantity will be indicated by the difference in the increased weight of the sulphate of barytes in the latter case. This may be regarded as an outline of the process. There are some details omitted, which will readily suggest themselves to the practical toxicologist.

In liquids containing organic matter.—If the sulphuric acid be mixed with such liquids as porter, coffee, or tea, the process for its detection is substantially the same, the liquid being rendered clear by filtration previously to adding the test. The sulphate of barytes, if mixed with organic matter, may be purified by boiling it in strong nitric acid; but this is not commonly necessary, as the reduction of the precipitate may be equally well performed with the impure, as with the pure sulphate. Some liquids generally contain sulphuric acid or a sulphate, such as vinegar and porter, but the acid is in very minute proportion; therefore, if there be an abundant precipitate, there can be no doubt, cæteris paribus, that free sulphuric acid has been added to them. Should the liquid be thick and viscid like gruel, it may be diluted with water, and then boiled with the addition of a little acetic acid. For the action of the test, it is not necessary that the liquid should be absolutely clear, provided it be not so thick as to interfere mechanically with the precipitation of the sulphate of barytes. So far with regard to articles administered, or of which the administration has been attempted.

Vomited matters.—These will commonly be found highly acid, reddening litmus paper, and causing effervescence with carbonated alkalies: they may be diluted with water, boiled, filtered and tested in the way above described. The acid is sometimes so intimately combined with decomposed mucus and blood, that it requires long boiling in order to separate it so that the barytic test may act readily. If the patient have been under treatment, these matters obtained from the stomach may have *no acid* reaction, either from the copious administration of water and abundant vomiting, or from an antidote having been used, such as magnesia. If on adding the test to the *neutral* liquid, there be a precipitate, sulphuric acid can be present only in the state of *sulphate*. If this precipitate be abundant, it cannot be due to the presence of minute traces of sulphates in the gastric and salivary secretions; but still it would be improper to infer from the chemical fact alone, that sulphuric acid had been swallowed, because it is well known that some saline sulphates, such as those of magnesia and soda, are often exhibited in large quantities medicinally, and it might be fairly objected to this evidence, that the precipitate was due to the presence of one of these salts. The symptoms, as well as other circumstances, would here aid the witness in forming an opinion—chemistry alone might mislead him.

In examining any organic liquid which has *no acid reaction*, it must be remembered that there are many salts in common use, some of them being medicines, which precipitate the barytic test. These are—all the soluble carbonates, iodates, phosphates, borates, tartrates, and oxalates. It is to be observed, however, that not one of these substances is precipitated by the test, provided the liquid for analysis be much diluted and acidulated with nitric acid before adding it. Should nitric acid alone produce any turbidness in an organic liquid, this may be again filtered and boiled before it is tested.

Contents of the Stomach.—When the patient survives, the analysis will of course be confined to the matters vomited. If the case proves fatal, we may, however, be required to examine the contents of the stomach. Should these be acid and give a precipitate with the test, it may be said that the acidity was due to the acids naturally contained in the gastric secretions (the muriatic and acetic,) which, however, are in very small proportion, or to some acid liquid, taken in the form of medicine or otherwise before death;—the precipitation by the test might also be ascribed to the presence of some medicinal sulphate. If the contents were not acid, then the effect produced by the test might be ascribed to the latter circumstance alone. All objections of this kind are at once removed not merely by resorting to the processes already described, but by noting particularly the presence or absence of the usual changes produced by mineral acids in the fauces, œsophagus, and stomach. The chemist might decide from an analysis alone; but the medical jurist should take into consideration the symptoms under which the deceased laboured, and the post-mortem appearances found in the body, before he ventures to pronounce an opinion from the results of his experiments.

Supposing the contents to give no evidence or but very slight evidence of the presence of the acid, we must then boil the altered or decomposed portions of the stomach in water for an hour, filter and apply the tests to the filtered liquid. But still no evidence of the presence of the poison may be obtained. Under these circumstances, it has been proposed by M. Taufflieb and Devergie to heat the stomach to a high temperature in a retort, the beak of which is plunged into a mixture of iodic acid and starch. (Ann. d'Hygiène, 1835, 1,427.) It is assumed that the non-discovery of the acid is due to its combination with the substance of the stomach in a way so intimate, that water cannot separate it. The application of heat therefore would, in the process above mentioned, lead to a decomposition of the sulphuric acid by the carbon of the animal matter, and its transformation to sulphurous acid. This would be immediately indicated by the production of the blue iodide of farina in the receiver. There are some objections which appear to me to render this process, thus applied, unfit for medico-legal purposes. Iodic acid is liable to be decomposed by many substances very different in their nature; as sulphuretted hydrogen gas, the hyposulphites and sulphurets, morphia, gallic acid, cyanide of potassium, sulphocyanide of potassium and the saliva, and it is not therefore safe to infer that the only deoxidizing agent in the distillation of the organic matter as above described, is the sulphurous acid, formed at the expense of the sulphuric acid, received *ab extra*, and combined with the tissues. Besides, mucous membrane contains sulphur, and on applying heat to it, to blood, serum, or other animal substances, sulphuretted hydrogen gas is evolved; this decomposes iodic acid, sets free iodine, and gives rise to error. The reaction is so extremely sensitive, that the very smallest portion of this gas will decompose iodic acid. (Med. Gaz. xxxvii. 954.) Hence it appears to me, that a medical jurist, when he finds no sulphuric acid in the stomach by the usual process of boiling the contents and the tissues for at least an hour, should rather declare that there is none present, than give an affirmative opinion of the existence of infinitesimal traces from the performance of a hazardous experiment.

It is a medico-legal fact of considerable importance, that the contents of the stomach in cases of poisoning by sulphuric acid, are often entirely free from any traces of this poison, even when it has been swallowed in large quantity. The acid is not commonly found when the individual has been under treatment, when there has been considerable vomiting, aided by the drinking of water or other simple liquids, or when the person has survived for a long period. If the case has been under treatment, the acid is either wholly absent or neutralized by antidotes. In support of this view, I might quote many reported cases; but I prefer giving two which I have witnessed. A girl swallowed four or five ounces of diluted vitriol, and died in eighteen hours. No portion of the acid could be detected in the stomach; but she had vomited considerably, and the acid was easily proved to exist in the vomited matters, by examining a portion of the sheet of a bed which had become wetted by them. In another case, nearly two ounces of the concentrated acid were swallowed; the patient died in twenty-five hours;—the stomach was most extensively acted on, and yet no trace of the acid could be discovered in the contents. The liquidity of the poison, and the facility with which it becomes mixed with other liquids, and ejected by vomiting, will readily furnish an explanation of this fact. In many cases of poisoning by sulphuric acid, therefore, a medical witness must be prepared to find, that chemical analysis will furnish only negative results. If the stomach should be perforated, the contents will be found in the abdomen, or perhaps in the lower part of the cavity of the pelvis:—they may then be absorbed by clean wetted linen or sponge, boiled with distilled water, and the solution examined for the acid in the way already described.

On solid organic substances.—It sometimes happens in cases of poisoning that sulphuric acid is spilled upon articles of clothing, such as cloth or linen, and here a medical jurist may succeed in detecting it, when every other source of chemical evidence fails. Again, sulphuric acid is often used for the purpose of seriously injuring a party, as by throwing it on the person,—an offence which, when accompanied with bodily injury, renders the offender liable to a severe punishment. On such occasions, proof of the nature of the corrosive liquid is required; and this is easily obtained by a chemical examination of part of the dress. The process of analysis is very simple. The piece of cloth should be digested in a small quantity of distilled water at a gentle heat, whereby a brownish-coloured liquid is commonly obtained on filtration. If sulphuric acid be present, the liquid will have a strong acid reaction, and produce the usual effects with the barytic test.

Stains on clothing.—These spots on clothing, if produced by the concentrated acid, are known in general, 1. by the black woollen cloth having its colour changed to a dirty brown, acquiring a red border after a few days. Diluted sulphuric acid produces at once, on black cloth, a red stain, which slowly becomes brown. 2. by their remaining damp or humid for a considerable period,—the sulphuric being a very fixed acid and readily absorbing water. That no objection may be offered to the result of an experiment of this kind, it is necessary that another part of the dress should be tested, in order to show that the sulphuric acid detected, is not due to the presence of any sulphate in the dress. Many articles of clothing, it must be remembered, yield slight traces of sulphates, when boiled in water. In the attempted erasure of writing from paper by diluted sulphuric acid, the same process will detect the presence of it. All white organic substances, such as calico or paper, although not blackened by diluted sulphuric acid, become, when impregnated with it, immediately charred on exposure to a moderate heat. The fibre of linen or cotton is slowly destroyed, even when the sulphuric acid forms only 1-30th part of the liquid. It may be objected to the medical evidence that this acid is used in bleaching cloth (*Queen* v. *Thomas*, Monmouth Lent Ass. 1847;) but the medical witness must bear in mind, that the cloth is also passed through a bath of chloride of lime and of alkali, so that all traces of free sulphuric acid are thereby removed. The colour of black leather is not changed by sulphuric acid.

In dyed articles of linen and cotton, the effect varies with the dye. In all cases the organic matter is sooner or later corroded and destroyed both by the concentrated and the weak acid. If the dress be dyed blue by indigo, the colour will be unchanged. (Nitric acid discharges the colour of indigo, turning it yellow.) If dyed with archil and some other blues, the spot may be reddened, and red streaks will be found wherever the acid vomited matter has passed over the dress. Logwood and madder, which are largely used in dyeing many of the common calicoes, are turned of a yellow colour by strong acids: and wherever an iron mordant has been employed in the pattern, there will be a rust red spot. In a very diluted state the acid is slow in acting, and the stain when recent is red. I have found by experiment, that whether the acid be used in a pure state, or darkened by organic matter, the effect is the same; and it would be impossible to say whether the colourless or coloured (carbonized) acid had been employed for the purpose of producing it. These stains, in cases of imputed poisoning, require very close chemical examination. In the case of Solomon Taylor (*Reg.* v. *Chesham*, Essex Lent Ass. 1847,) the prisoner was charged with the murder by poison of an infant, by thrusting some corrosive substance into its mouth. The witnesses for the prosecution deposed that they saw the prisoner put something of a pink colour into the child's mouth; that the child was then sick, and vomited over its own dress, some of the liquid falling upon an apron which the prisoner wore. There was no proof of poisoning, but these spots were considered to bear strongly against the prisoner. On examining them, I found that the dress of the child had been dyed with cochineal pink; that the stains were of a *crimson colour, and not corroded*:—the apron worn by the prisoner was dyed with madder-purple; the spots on it were small and circular, not lengthened, like those which would be produced by the act of vomiting; the dye was changed to a *yellow*, and the fibre corroded. It was therefore clear that the spots on the child's dress had been caused by an *alkali;* those on the apron by an *acid*. They could not have been produced at the same time and from the same cause. The prisoner was acquitted.

I have ascertained by experiment, that sulphuric acid may be easily detected on articles of clothing after many years exposure. In January 1831, a small quantity of this acid was spilled on a black cloth dress: it has been exposed in an open jar to the air for upwards of *sixteen years*. The cloth is changed to a deep brownish-black colour; it is soft, and yields to water a brownish coloured acid liquid, in which a large quantity of sulphuric acid may be still easily detected. In a case of poisoning, which occurred in 1832, the acid was partly spilled on a dress of printed cotton. This has been likewise exposed for fifteen years:—the organic fibre is completely corroded by the acid, and reduced to a kind of humid powder; from this substance, by the addition of water, a liquid is obtained, the acidity of which is proved by the barytic test to be due to the presence of sulphuric acid. These facts are of some interest, because it has been generally supposed, that the stains on clothing soon lose all traces of the acid, partly by decomposition in contact with organic matter, and partly by evaporation; but it is hereby evident, that such stains, if not removed by washing, may be, in some instances, detected for a period of time much longer than is ever likely to be required in any medico-legal investigation.

In determining the presence of sulphuric acid in certain articles of clothing, I have found a modification of the iodic acid test extremely delicate and easy of application. The following is the way in which it may be applied:—About half a grain of the article of dress (cotton,) impregnated with sulphuric acid, is introduced into a short piece of glass tube, about one-eighth of an inch in diameter, and closed at one end. The stuff is then gently heated, and at the same time a piece of paper, previously saturated with starch and moistened with a drop of iodic acid, is brought near to the mouth of the tube. The blue iodide of farina is immediately produced by the sulphurous acid formed at the expense of the sulphuric acid, locked up in the organic substance. This process is only strictly applicable to the examination of those articles of dress which contain *no sulphur*, as cotton or linen, or which are not impregnated with compounds containing sulphur; thus the presence of serum, blood, or mucus, owing to these substances containing sulphur, would lead to fallacious results. All kinds of flannel and woollen-cloth contain sulphur as a natural constituent: hence, whether sulphuric acid be present or not, they will decompose iodic acid by heat. It is highly necessary to bear in mind this fact, as the following case will show. In April 1846, Mr. Eastes of Folkestone brought to me for examination the stomach of a man who, it was supposed, had been poisoned by sulphuric acid (ante, p. 61.) There was a dark coagulum of blood and mucus lining the stomach, and the surface of the mucous membrane was reddened; but the most careful analysis showed that not a particle of sulphuric acid was present. A portion of the string with which the stomach was tied (which was of a dark colour,) was dried and heated in a tube. The vapour immediately decomposed iodic acid, which I attributed to its having been impregnated with part of the dark coagulum of blood and mucus, as no sulphuric acid was found in it, and an unstained portion of string produced no effect on iodic acid. (Med. Gaz. xxxvii. 954.) In order to obviate any fallacy in the employment of this test, the analyst should always apply the test to an unstained portion of the same cloth, whether linen or cotton. We can never trust to the results, in the analysis of any kind of woollen cloth or silk.

AROMATIC SULPHURIC ACID.

There is a preparation under this name in the Pharmacopœias of Edinburgh and Dublin; it is also known as elixir of vitriol, or acid elixir of vitriol.

It is a mixture of sulphuric acid and rectified spirit (sulpho-vinic acid,) to which ginger and cinnamon are added. According to the Dublin College, the sulphuric acid forms about one-tenth by measure. It is therefore a diluted form of sulphuric acid.

[This preparation is known in the United States as elixir of vitriol. According to the United States Pharmacopœia, the acid is to the alcohol as 1 to 4.15 by weight. The Dublin article contains 1 of acid to 5.33 of alcohol by measure.—G.]

SYMPTOMS.

One instance of poisoning by it has been reported by Mr. Blyth. A woman, æt. 25, swallowed *ten drachms* of the acid in mistake for a black draught. She experienced a sharp, burning, and disagreeable taste, great heat and pain in the stomach and œsophagus, accompanied with constant vomiting of a dark-coloured liquid streaked with blood, with great difficulty of deglutition. Magnesia and water were freely given, so that before medical assistance had arrived, the acid was expelled from the stomach. In about eleven hours there was a considerable discharge of blood from the rectum. There was irritability of the stomach, with thirst, and a copious discharge of saliva, but in two days the patient perfectly recovered. (Med. Gaz. xxv. 944.)

ANALYSIS.—The acid may be precipitated by the addition of carbonate of barytes: the precipitate, washed with nitric acid, and the residue calcined with charcoal, would indicate the presence of a sulphate by the production of a sulphuret. Alcohol might be separated by distillation, with or without the previous addition of carbonate of potash.

QUANTITATIVE ANALYSIS OF SULPHURIC ACID.

It may be sometimes necessary to state how much sulphuric acid is present in a particular liquid. In order to determine this point, a portion of the liquid should be measured off, and the whole of the sulphuric acid present precipitated by the salt of barytes. The sulphate of barytes should be rendered pure by boiling it in nitric acid, then washed, dried, and weighed. For every one hundred grains of dried sulphate obtained, we must allow half the weight, *i. e.* fifty grains of common oil of vitriol (bihydrate) to have been present: hence the rule is a very simple one. As the equivalent of the bihydrated acid is 58, and that of sulphate of barytes 116, the proportion of acid is one-half the weight of the precipitate. If we thus obtain the weight of the sulphuric acid present, it is very easy, from its known specific gravity, to calculate the quantity by *measure*. It is important for the analyst to remember, that the specific gravity of sulphuric acid is nearly twice that of water, being equal to 1.84: hence one ounce by measure will be nearly equal to two ounces by weight. I have found that one fluid-drachm of common sulphuric acid weighs 119 grains; and one fluid-ounce, measured in the same measure, weighed 932 grains,—a difference of 20 grains, which was probably due to the irregular division of the glass.

SULPHATE OF INDIGO.

Several cases of accidental poisoning by this substance have occurred. As the compound is nothing more than a solution of indigo in common sulphuric acid, the symptoms and post-mortem appearances are the same as those that have been described for the latter substance. This kind of poisoning may be suspected, when, with these symptoms, the membrane of the mouth has a blue colour. The vomited matters, as well as the fæces, are at first of a deep blue tint; afterwards green; and it was observed in two instances that the urine had a blue tinge. One of these, reported by Orfila, was the case of a child, which died in seven and a half hours. The other was observed by M. Bouchardat, and is of some interest. A young woman, aged 18, swallowed—as it was conjectured—about *an ounce* of the sulphate of indigo. *Immediately* afterwards she felt an acute burning pain in the throat and in the stomach. She threw herself on the ground, and her cries soon brought around her her neighbours, who found her vomiting a bluish coloured liquid, which effervesced on the pavement. A quantity of oil and milk was immediately exhibited; the milk was speedily thrown up coagulated, and of a *blue* colour. When brought to the hospital, three hours afterwards, she was in the following condition: her face pale; features somewhat altered; her eyes were sunk, and her lips of a violet tinge. There was a yellowish-coloured spot on the upper lip, at each angle of the mouth. The tongue was blue, the throat was painful, and there was a sense of constriction. The epigastrium was tender. There was no pain in the abdomen; obstinate constipation; respiration difficult; great anxiety; coldness of the upper extremities, and a quick and small pulse. Her intellect was clear, and her answers to the questions put, were sensible and proper. Four drachms of calcined magnesia were administered in a pint of water: much of this was rejected by vomiting, accompanied by bluish clots. A few hours afterwards the pain in the throat became very severe, the upper extremities cold, and the pulse imperceptible. The urine which she passed had a *slight tinge of blue*. She continued to become worse; the vomiting of chocolate-coloured matter returned; and she died about ten o'clock—*i. e.* about eleven hours after having taken the poison.

The body was examined 27 hours after death. The head presented no particular appearance. There was no sign of corrosion in the mouth. The mucous membrane of the pharynx and œsophagus became easily detached in dry, white, brittle layers. The heart was filled with three ounces of *coagulated blood;* the aorta was also filled with brown and semi-liquid clots; the lining membrane of this vessel was of a bright red colour! The stomach was distended, containing two ounces of a brown coloured liquid. The mucous membrane was carbonized, and of the colour of soot, with slight patches of redness throughout its whole extent, except for about an inch near the pylorus, where it was of a rose-red colour. It was easily detached in layers; but there was no trace of ulceration. The membrane of the duodenum was inflamed and ulcerated, and in parts it was found corroded and blackened. A dark-coloured mucus was seen in the small intestines, and patches of a blue colour were scattered through the colon. The femoral arteries were filled with a semi-coagulated dark-coloured blood. The cavity of the left femoral artery was completely obstructed by the clot.

M. Bouchardat, who reports this case, considers that the deceased died from the absorption of the acid into the blood-vessels,—by which the blood was coagulated, and the circulation arrested. Reasons have been already assigned which appear to me to render this theory improbable (ante, p. 168.) Several instances of recovery are on record. Dr. Galtier reports two,—one of which is the case of a young woman, who swallowed rather more than an ounce of sulphate of indigo. She recovered on the eighth day. Calcined magnesia and milk were found to be the best remedies. (Toxicologie, i. 206.)

ANALYSIS.—The process is the same as that described for sulphuric acid in organic mixtures. The blue colour of the sulphate is immediately destroyed by boiling it with nitric acid. The barytic test may then be employed in the usual way.

CHAPTER XVI.

POISONING BY NITRIC ACID OR AQUA FORTIS. ACTION OF THE CONCENTRATED AND DILUTED ACID—DESTRUCTION OF THE STOMACH—POST-MORTEM APPEARANCES—QUANTITY REQUIRED TO DESTROY LIFE—PERIOD AT WHICH DEATH TAKES PLACE. PROCESSES FOR DETECTING THE POISON IN PURE AND ORGANIC LIQUIDS—ON ARTICLES OF CLOTHING.

General Remarks.—This substance is popularly known under the name of Aqua fortis, or Red spirit of nitre. According to Tartra, it seems to have been first used as a poison about the middle of the fifteenth century. Although it is perhaps much more used in the arts than oil of vitriol, cases of poisoning by it are by no means so common. Tartra was only able to collect fifty-six cases extending over a period of nearly four hundred years; and it appears from the return of inquisitions for 1837-8, there were only two instances reported to have occurred in England during those two years. Cases of poisoning by this acid have been chiefly the result of accident or suicide. I have only met with one instance where it was poured down the throat of a child for the purpose of murder. The *external* application of nitric acid has been a criminal cause of death on several occasions:—in one instance the acid was poured into the ear of a person while sleeping, and it led to the slow destruction of life. These are not strictly cases of poisoning, but more nearly approximate to death from wounding or mechanical violence.

SYMPTOMS.

THE CONCENTRATED ACID.—These, on the whole, bear a close analogy to those produced by sulphuric acid. They come on *immediately*, and the swallowing of the acid is accompanied by the most intense burning pain in the fauces and œsophagus, extending downwards to the stomach:—there are gaseous eructations, from the chemical action of the poison,—swelling of the abdomen, violent vomiting of liquid or solid matters, mixed with altered blood of a dark brown colour, and shreds of mucus, having a strong acid reaction. The abdomen is generally exquisitely tender; but in one well-marked case of poisoning by this acid, the pain was chiefly confined to the fauces: probably the poison had not reached the stomach. The mucous membrane of the mouth is commonly soft and white, after a time becoming yellow, or even brown; the teeth are also white, and the enamel is partially destroyed by the chemical action of the acid. There is great difficulty of speaking, as well as of deglutition, the mouth being filled with viscid mucus: the power of swallowing is sometimes entirely lost. On opening the mouth, the tongue may be found swollen and of a citron colour; the tonsils are also swollen and enlarged. The difficulty of respiration is occasionally such, as to render tracheotomy indispensable, especially in young subjects. (Case by Mr. Arnott, Med. Gaz. xii. 220.) As the symptoms progress, the pulse becomes small, frequent, and irregular; the surface of the body extremely cold, and there are frequent rigors. The administration of remedies—even the deglutition of the smallest quantity of liquid, increases the severity of the pain, occasions vomiting, and gives rise to a feeling of laceration or corrosion. (Tartra, 144.) There is obstinate constipation. Death takes place in from eighteen to twenty-four hours, and is sometimes preceded by a kind of stupor from which the patient is easily roused. The intellectual faculties commonly remain clear until the last. In one instance the patient was insensible, but she ultimately recovered. Death may be occasioned by this acid, in consequence of its action on the larynx, as in the case of sulphuric acid. Should the patient survive the first effects of the poison, the mucous membrane of the fauces and œsophagus may be discharged, either in irregular masses, or in the form of a complete cylinder of the œsophageal lining. There is great irritability of the stomach, with frequent vomiting and destruction of the powers of digestion: the patient becomes slowly emaciated, and dies from starvation or from exhaustion.

THE DILUTED ACID.—The symptoms above described apply to acute cases of poisoning by concentrated nitric acid. When the acid is *diluted*, they are somewhat modified according to the degree of dilution. A remarkably interesting case of poisoning by diluted nitric acid has been lately published by Dr. Puchelt, of Heidelberg, which not only shows the progress of the symptoms, but also the powers of nature in resisting for a time the chemical destruction of an important organ. A man, aged 52, swallowed two ounces of diluted nitric acid (the strength not stated.) He was *immediately* seized with severe burning pain in the mouth, fauces and œsophagus: this was followed by vomiting, whereby the greater part of the acid was probably ejected. He was not seen for several hours, and then the symptoms had so far subsided that the hospital assistant sent him away as not requiring immediate attendance. An oily emulsion was subsequently given to him. After the lapse of thirty-six hours, he was admitted into the hospital, and was for the first time seen by Dr. Puchelt. The mucous lining of the mouth and pharynx was covered with a white shreddy membrane which could be readily peeled off: parts were already abraded. There were yellow stains on the cuticle around the mouth, especially upon the upper lip. The patient experienced great difficulty in swallowing: respiration was laborious, the epigastrium tender, the abdominal parietes hard and retracted. On the whole, the symptoms were very favourable, and led to the suspicion that but little injury had been done to the stomach. Leeches and other antiphlogistic means were employed, and in about eight days he began to retain a portion of food on the stomach. Nevertheless, his strength diminished, and he became emaciated: on the fifteenth day the food which he took was rejected; on the sixteenth some blood was found mixed with the stools; on the seventeenth there was great pain, with vomiting of black fluid blood, and of decomposed membrane of a fibrous structure, which when spread out, was a foot in breadth. This membrane was marked with black spots, as if it were burnt and perforated with numerous small and large apertures. A large quantity of black putrid blood was at the same time passed by stool. The symptoms became after this more unfavourable, and the vomiting of blood frequently recurred, until death took place on the twenty-third day after the poison had been swallowed. On opening the abdomen, there was no appearance of a stomach, but in its place a cavity formed by the liver, colon, and other viscera: the interior wall, lesser curvature, and upper part of the posterior wall, being wholly absent. A dark green mass was spread over the interior: but the parietes were so soft as to give way on the slightest pressure. The intestinal canal, with the exception that it contained a large quantity of bloody matter, presented nothing peculiar. The mucous membrane of the œsophagus was found removed throughout its whole length. (Ein Fall von Vergiftung mit Scheidewasser, von Dr. F. A. B. Puchelt, Heidelberg, 1845.)

There is no doubt that the diluted acid was in this case much stronger than that of the English Pharmacopœia, which contains one-tenth by measure of strong nitric acid; or one part of nitric acid to nine parts of water. I have not met with any instance of poisoning by this diluted acid.

POST-MORTEM APPEARANCES.

A full account of these will be found in the well-known work of Tartra (Essai sur l'Empoisonnement par l'Acide Nitrique,) published upwards of forty years ago. (An. x.)

Supposing death to have taken place rapidly, the following appearances will be met with. The skin of the mouth and lips will present various shades of colour from an orange-yellow to a brown; it appears like the skin after a blister or burn, and is easily detached from the subjacent parts. Yellow spots produced by the spilling of the acid, may be found about the hands and neck. A yellow frothy liquid escapes from the nose and mouth, and the abdomen is often much distended. The membrane lining the mouth is sometimes white, at others of a citron colour; the teeth are white, but present a yellowish colour about the coronæ. The pharynx and larynx are much inflamed; the latter sometimes œdematous. The lining membrane of the œsophagus is softened, and of a yellow or brown colour, easily detached, often in long folds. The trachea is more vascular than usual, and the lungs are congested. The most strongly marked changes are, however, seen in the stomach. When not perforated, this organ may be found distended with gas—its mucous membrane partially inflamed with patches of a yellow, brown, or green colour, or it may be even black. This green colour is due to the action of the acid on the colouring matter of the bile; but it must be remembered that a morbid state of the bile itself often gives this appearance to the mucous membrane in many cases of death from natural disease. There is occasionally inflammation of the peritoneum, and the stomach is glued to the surrounding organs. Its coats are often so much softened, as to break down under the slightest pressure. In the duodenum similar changes are found; but in some cases the small intestines have presented no other appearance than that of slight vascularity. It might be supposed that the stomach would be in general perforated by this very corrosive substance; but this is far from being the case. Tartra only met with two instances, and in one of these, the individual survived twenty, and in the other thirty hours. In giving this poison to rabbits, I have not found the stomach perforated, although the acid had evidently reached that organ, from its coats being stained of a deep yellow colour. In these experiments the non-perforation appeared to be due to the protective influence of the food with which the stomach was distended. In the very few cases that are reported in English journals, it would appear that the stomach has not been perforated: the poison had been swallowed soon after a meal, and its parietes had thus escaped the corrosive action of the acid.

In cases of *chronic* poisoning, *i. e.* where death takes place *slowly*, the appearances are of course very different, as the following case will show. A man, aged thirty-four, swallowed a wine-glassful of nitric acid, but the greater portion was immediately rejected by vomiting. An attack of acute gastritis followed, which was combatted by the usual remedies. The man was discharged from the hospital into which he had been admitted, in three weeks; but about a month afterwards, he was readmitted, in consequence of his suffering severe pain extending down the œsophagus to the epigastrium, as well as from vomiting after taking food. The patient gradually sank, and died three months after he had taken the acid. On dissection the pylorus was found so diminished in size, that its diameter did not exceed a line or two, and the duodenum was equally contracted for about an inch and a half from its commencement. The mucous membrane was softened and red in patches; and there were several cicatrices of ulcers. The subjacent tissues were in a scirrhous state. (See Med. Chir. Rev. vol. xxviii. 553.) As a contrast to this, the appearances met with in a case of poisoning by nitric acid, which proved *rapidly* fatal, may be here described. A man swallowed about two ounces of aqua fortis, and died speedily from its effects. On examining the body, the lips were found partly yellow and partly of a brownish-red colour, dried up like parchment. Several yellow or parchment-coloured spots were observed on the chin, as also on the cravat. The mucous membrane of the mouth was white and easily detached,—that of the tongue was dry and hard,—that of the pharynx and œsophagus yellowish-green in colour and of a leathery consistency. The stomach contained a dark-coloured liquid, highly acid. It was externally mottled of a greenish-blue and black colour. The mucous membrane throughout was softened and in a gangrenous state. The same appearances were met with, although in a less degree, in the duodenum and upper part of the ileum. On analysis, the contents of the stomach yielded nitric acid. (Von Raimann Medicinisch. Jahrb. 20 B. 2 S. p. 221.)

QUANTITY REQUIRED TO DESTROY LIFE.

The remarks made on this subject in speaking of sulphuric acid, apply here. Tartra states, that the quantity usually taken varies from one or two drachms to four ounces,—never more than this; but in most of the cases which he reports, the quantity taken is not mentioned. Indeed, the obtaining of any information of this kind is purely accidental; and the determination of the exact quantity swallowed, must be therefore very difficult. One point is certain;—the same quantity will not kill two individuals in the same time,—one may die slowly, and the other rapidly, according to whether the stomach at the time contains food or not. The *smallest* quantity which I find reported to have destroyed life, is about *two drachms*. It was in the case of a boy, aged thirteen: he died in about thirty-six hours. But less than this, even one drachm, would doubtless suffice to kill a child; and, under certain circumstances, an adult; for the fatal result depends on the extent of the mischief produced by this corrosive poison in the larynx, œsophagus, and stomach. What is the largest dose of concentrated acid from the effects of which a person has recovered, it is difficult to say: since in most of the cases of recovery mentioned by authors, the quantity of the poison taken, was unknown.

In one instance recently reported, a woman (æt. 26) recovered in a few days after having swallowed *half an ounce* of aqua fortis of the usual strength. There was great reason to believe that the poison did not reach the stomach, or that it had produced but little action on the organ. The chief seat of pain was in the fauces and œsophagus. (Lancet, May 8, 1847, p. 489.)

PERIOD AT WHICH DEATH TAKES PLACE.

This must depend on the quantity swallowed, the strength of the acid, and whether any medical treatment has or has not been adopted. Out of twenty-seven deaths from nitric acid, reported by Tartra, in nineteen it destroyed life rapidly, and in eight slowly. This author met with two instances in which death took place within *six* hours after the poison was swallowed; but he considers that the greater number who fall victims to the direct effects of the acid, die within twenty-four hours. Sobernheim relates a case of poisoning by nitric acid, which proved fatal in *one hour and three quarters*. (Op. cit. 402.) This I believe to be the *most rapidly* fatal case on record, where the acid acted as a poison. The usual well-marked effects were found in the œsophagus, stomach and duodenum. In young infants, however, life may be destroyed by this poison in a few minutes, should it happen to affect the larynx. A woman shortly after her delivery, in the absence of her attendants, poured a quantity of nitric acid into the mouth of her young infant. The mother concealed from those about her this attempt at murder; but medical assistance was immediately sent for. The child died in a *few minutes*. Some of the acid had been spilled; and from the yellow colour of the stains, the medical man suspected that the child had been poisoned by aqua fortis. On inspection, nitric acid was found in its stomach, and the mother confessed the crime. (Cazauvieilh, Du Suicide et de l'Aliénation Mentale, p. 274.) Although in this case no mention is made of the state of the fauces and larynx, it is highly probable, from the rapidity with which death took place, that this event was in great part due to suffocation. The following experiment will perhaps serve to show how speedily life may be destroyed under these circumstances. Half a drachm of concentrated nitric acid was given to a rabbit. In about half a minute, it became insensible, and apparently lifeless. There was no sign of pain or irritation. It died in *one minute*. A small quantity of gaseous matter, in the form of a fuming vapour, escaped from the nostrils. The abdomen became much swollen before death. On inspection, it was found that the poison had strongly acted on and corroded the parts about the larynx. A portion had penetrated into the lungs, turning them yellow, and corroding them. Another portion had been swallowed, and had entered the stomach, producing the usual yellow stains, but it had not perforated the organ. With regard to the *longest* period at which death has taken place from the effects of this poison, a case has been already related, where a man who had swallowed nearly two ounces, did not die until three months afterwards. The longest case is perhaps that recorded by Tartra, where a woman perished from exhaustion, produced by the secondary effects of the poison, *eight months* after having swallowed it.

TREATMENT.

It may be the same as that recommended in poisoning by sulphuric acid. In addition to the remedies there suggested, a diluted solution of carbonate of soda with barley-water, and other demulcents, may be administered. In many cases, there is an utter impossibility of swallowing even the smallest quantities of liquid: and if an attempt be made to introduce these remedies by a tube, there is great risk of perforating the softened parietes of the pharynx, larynx, or œsophagus. Should suffocation be threatened, then tracheotomy may be resorted to. Modern experience is rather against the recovery of these cases, under any form of treatment:—but according to Tartra, in accidental poisoning by this acid, there is very great hope of recovery, if the case be timely attended to. He states that out of thirty-one such cases, twenty-three recovered, seventeen perfectly; while out of twenty-four cases, wherein suicide was attempted, only six recovered. (Op. cit. p. 186.)

CHEMICAL ANALYSIS.

In the simple state. This acid may be met with either concentrated or diluted. The *concentrated acid* varies in colour from a deep orange red to a light straw yellow. It may be recognised—1. By evolving acid fumes when exposed.—2. By its staining organic matter yellow or brown, the colour being heightened and turned of a reddish tint by contact with caustic alkalies.—3. When mixed with a few copper cuttings, it is rapidly decomposed—a deep red acid vapour is given off, and a greenish coloured solution of nitrate of copper is formed. Tin or mercury may be substituted for copper in this experiment.

TESTS.—In the *diluted* state. This acid is not precipitated like the sulphuric by any common reagent, since all its alkaline combinations are soluble in water. —1. The liquid has a highly acid reaction, and on boiling it with some copper turnings, red fumes of nitrous acid vapour are given off, unless the proportion of water be very great. At the same time, the liquid acquires a blue colour.—2. A streak made on white paper with the diluted acid, does not carbonize it when heated; but a scarcely visible yellow stain is left.—3. The liquid is neither precipitated by nitrate cf barytes nor by nitrate of silver. These two last experiments give merely negative results—they serve to show that the sulphuric and muriatic acids are absent.—4. By dipping a piece of bibulous paper in a weak solution of potash, and then in a portion of the acid liquid, and drying it, it will be found on igniting it, if the acid be the nitric, that the paper burns with deflagration. This is not a property peculiar to nitric acid; but it distinguishes it from any of the common acids used as poisons. The chlorates, bromates, iodates, permanganates and bichromates, burn with deflagration in contact with organic matter. I have also observed this with respect to one metallic oxalate—the oxalate of silver.

In order to detect nitric acid, the liquid should be carefully neutralized by potash, and then evaporated slowly to obtain crystals. If the liquid contain nitric acid, these crystals will possess the following characters;—1. They appear in the form of lengthened fluted prisms, which neither effloresce nor deliquesce on exposure. One drop of the solution evaporated spontaneously on glass will suffice to yield distinct and well-formed crystals. This character distinguishes the *nitrate* of potash from a very large number of salts. When neutralized with soda, the crystal is of a rhombic form,—a very striking character of *nitrate* of soda.—2. When reduced to powder and heated with their bulk of finely powdered charcoal on platina-foil, the mass suddenly ignites and burns with the well-known phenomena of deflagration. There are other salts that possess this property when heated with charcoal (suprà.)—3. When moistened with strong sulphuric acid, the powdered crystals slowly evolve a colourless acid vapour. By this test, the nitrate is known from every other deflagrating salt.—4. A portion of the powdered crystals should be placed in a small tube and mixed with their bulk of fine *copper* filings. The mass is then to be moistened with water, and a few drops of strong *sulphuric acid* added. Either with or without the application of a gentle heat, a decomposition immediately ensues, by which red fumes of *nitrous acid* are evolved, recognisable by their colour, odour and acid reaction. This test answers equally well with a nearly saturated solution of the salt in water, It need hardly be observed that the sulphuric acid employed in all these experiments should be free from nitric acid.

This last test is conclusive, and renders it unnecessary to resort to any other experiment. It is so delicate, that, by using a tube only one-eighth of an inch in the bore, one-tenth of a grain of nitrate of potash will give very satisfactory results. This is equivalent to about one-twentieth of a grain of nitric acid.—a quantity to which the toxicologist will not often have to confine his analysis in medico-legal practice. I have found it advantageous in these cases to adopt the following method of employing the test. Wrap the minute portion of nitrate in a small piece of the finest copper gauze, and drop the pellet thus formed to the bottom of the small tube. One drop of water followed by a drop of strong sulphuric acid, will, by the aid of heat, suffice to evolve the red fumes of nitrous acid. Many other tests have been proposed for the detection of nitric acid as a nitrate.—1. The boiling of the suspected liquid with a small quantity of diluted sulphate of indigo, when if nitric acid or a nitrate be present, the colour is discharged, but the same result takes place with iodic acid.—2. The boiling of the suspected nitric acid or solution of nitrate, with a few drops of muriatic acid and gold leaf, when the gold will become dissolved,—a fact made evident if not by the entire disappearance of the metal, by adding to the liquid a solution of chloride of tin. A mixture of muriatic with the iodic acid will also dissolve gold. —3. Add to the liquid, a crystal of green sulphate of iron; if nitric acid be present, a dark green liquid is formed around the crystal as it dissolves:—if the solution be suspected to contain a nitrate, a few drops of sulphuric acid must be added, when the same effects will follow. When the quantity of nitrate is exceedingly small (i. e. 1-5000th.) the modification of this test, suggested to me by Mr. Schweitzer, will be found serviceable. A drop of sulphuric acid should be placed in a reduction-tube: one or two drops of water added without mixing, then a minute crystal of green sulphate of iron—lastly, the suspected salt (the 1-20th of a grain of nitrate of potash.) A pink or dark-coloured stratum of liquid will appear about the edge of the crystal. I have found this a very delicate test, and it distinguishes a nitrate from a chlorate; but sulphate of iron decomposes both iodic acid and an iodate, and a pink-coloured film was, according to my experiments, equally formed in these cases. The gas produced by the action of sulphuric acid on a nitrate when mixed with copper, may be conducted by a tube into a solution of green sulphate of iron.—4. Add to the suspected salt in powder, a few grains of morphia and some strong sulphuric acid:—if it be a nitrate, the saline mass acquires an orange or a yellow-brown colour, from the action of the evolved nitric acid on the morphia (O'Shaughnessy.) The results in this case are certainly not so uniform or satisfactory as those obtained by the action of sulphuric acid and copper filings.—5. Orfila has lately proposed the sulphate of narcotine as a test for nitric acid or a nitrate (i. 137.) If strong sulphuric acid be poured on narcotine, the latter acquires a gamboge-yellow colour, which, when the twentieth part of a grain or less of strong nitric acid or a nitrate is added, is changed to a deep blood-red, becoming more intense after a time. According to the same authority the sulphate of brucia is even more sensitive, the alkaloid acquiring a blood-red colour under the same circumstances; but I do not find the result to be so satisfactory. The sulphuric acid should in either case be added to the alkaloid first, since it might itself contain nitric acid as an impurity, and thus lead to error. The objections to the test are, that change of colour does not occur with diluted nitric acid, and if the nitre be at all impure from organic matter, the results are rendered obscure. A very similar change of colour is also produced on adding iodic acid, an iodate, or a chlorate to sulphate of narcotine. The most satisfactory test is the decomposition by copper and sulphuric acid. (p. 188.)

Objections.—I know of no objections which can be urged to the mode of testing for nitric acid above recommended. The process of deflagration is merely one of a series of characters, and cannot of itself furnish evidence of the presence of a nitrate. It may be regarded simply as a trial-test. With respect to the action of copper filings and sulphuric acid, there is no salt of potash but the nitrate (that which is compounded of nitric acid) which gives such results as have been described. The bromide of potassium gives off ruddy vapours (bromine,) somewhat resembling those of nitrous acid; but these bleach litmus paper, and are in other respects easily distinguishable. Besides, these ruddy vapours are given off from the solid bromine by sulphuric acid without the addition of copper filings, whereas a nitrate under similar circumstances yields a colourless acid vapour. It may be safely affirmed, that bromine could not be mistaken for nitrous acid vapour, by one who had been at all accustomed to chemical analysis.

In acid liquids containing organic matter.—Nitric acid may be administered in such liquids as vinegar or porter. In this case, besides the acid reaction, there will be the peculiar smell produced by the acid, when mixed with substances of an organic nature. The application of the usual tests is here counteracted:—thus unless the quantity of nitric acid in such a liquid as porter be very considerable, the orange-red fumes of nitrous acid are not evolved on boiling the liquid with copper-cuttings. If the liquid be viscid, this viscidity must be destroyed by dilution with water:—and in all cases, if any solid or insoluble substances are floating in it, as in the *matters vomited* or *contents of* *the stomach*, it must be filtered, in order to obtain at least a portion of the acid liquid. This operation is commonly very slow. If we procure the clear acid liquid, the colour may be disregarded. We should then carefully neutralize it with a weak solution of potash, or its carbonate; and boil it with a large quantity of well-washed animal charcoal for two hours. On filtration, it will probably come through of a pale yellow colour. If the colour be at all deep, it must be reboiled with a fresh quantity of animal charcoal, and now on filtration it will be tolerably clear. Concentrate to a small bulk by evaporation. As a trial-test we may dip in a slip of bibulous paper, dry it, and observe whether it burns with deflagration. This commonly answers, unless the quantity of nitric acid present be very small, or unless the nitrate of potash formed, be mixed with a large portion of some other salt. A few drops of the liquid may be crystallized on a piece of glass, by slow evaporation; and the resulting crystals examined for all those properties which have been described as characteristic of the compound of potash with nitric acid. The crystals obtained, may be coloured and impure. This circumstance does not at all interfere with the action of the most important test for nitric acid, namely, that by copper-filings and sulphuric acid. They may, however, if necessary, be purified by digesting them in pure ether, or pure alcohol. These liquids do not dissolve the nitrate of potash, but will often serve to remove from it the organic matters by which it is coloured. This process, according to my observation, is very effectual in detecting nitric acid, when mixed with liquids resembling porter. If the acid has been administered in vinegar, acetate of potash, as well as nitrate, is formed during the neutralization. The acetate may be separated by pure alcohol, in which it is very soluble, while the nitrate remains undissolved. Other modifications will suggest themselves, according to the nature of the liquid with which the acid is mixed.

Neutral liquids.—But the vomited matters and the contents of the stomach may have no acid reaction, and yet nitric acid be present. Thus it may have become neutralized by lime or magnesia, through the administration of antidotes. In such a case, it would not be easily detected, unless it were in pretty large quantity. By the addition of carbonate of potash to the filtered neutral liquid, these earthy nitrates may be transformed to nitre, and the analysis then proceeded with,—the carbonates of lime and magnesia formed, being separated by filtration. Again, for the same reasons as those mentioned in speaking of sulphuric acid, the liquid found in the stomach of a person who has died from nitric acid, may not contain a trace of the poison, either free or combined. Its absence, therefore, does not negative a charge of poisoning. Nitric acid has a much stronger tendency than the sulphuric to combine with the solid organic tissues; and in decomposing them, it undergoes decomposition itself. In a case of this kind, those parts of the mucous membrane, whether of the œsophagus or stomach, which are stained yellow or corroded, should be boiled in water previously rendered alkaline by potash. The resulting liquid may then be examined for nitre. This experiment, from the small quantity of free acid present, is very likely to fail. In examining the stomachs of rabbits killed by nitric acid, I have found that even deeply stained portions of the mucous membrane have yielded commonly only very faint traces of acid. But the discovery of no more than traces of acid in these cases of poisoning, is, in my opinion, tantamount to a failure of the chemical branch of evidence; for no inference could be drawn from such minute results relative to the fact of poisoning, unless the evidence from symptoms and post-mortem appearances, with moral circumstances, were sufficiently conclusive; and when this is the case, whether the poison be wholly absent, or exist only in infinitesimal traces, must be a matter of trifling importance. It may perhaps be proper to mention in this place, that the nitrates have not been found to exist as natural constituents of the secretions of the alimentary canal. If any alkaline chloride be present in the organic liquid, it will be difficult to obtain evidence of nitric acid or a nitrate by the usual tests, since a decomposition would ensue whereby chlorine would be evolved. In order to counteract this difficulty, Dr. Christison has recommended the entire precipitation of the chloride in the first instance, by a hot solution of acetate of silver. Oxide of silver would answer the same purpose. Orfila states that he has discovered nitric acid in the urine of animals poisoned by it. He considered that it had been absorbed and carried into that liquid. (Op. cit. i. 145.)

On solid organic substances.—We have already spoken of the modification required in the analysis, in reference to the supposed presence of the poison in the discoloured or corroded portions of the mucous membrane. This acid is, however, sometimes maliciously thrown at persons; and we may be required to examine some articles of dress, suspected to have been stained by it. The spots produced by strong nitric acid on woollen stuffs, are either of a yellow, orange-red, or a brown colour, according to the time at which they are seen. On black cloth they are at first of a light brown colour, passing after eight or ten days to an olive green with a red border. After a time they become dry, (unlike those produced by strong sulphuric acid,) and the texture of the cloth is entirely destroyed. In order to examine them, the stained portions may be cut out and digested in a small quantity of warm distilled water. If nitric acid be still present, the water will acquire an acid reaction; but in order to establish this, the liquid must be neutralized by potash, then evaporated, and examined for nitrate of potash in the way already described. Should the water acquire no acid reaction, then there is no perceptible quantity of acid present. To render this certain, however, the water may be made feebly alkaline by potash, and again boiled with the stuff; the liquid may be filtered and examined for nitre. It is rare that any evidence of the presence of nitric acid is obtained by this latter process, when the stained portions of cloth do not give out any free acid to the distilled water in the first instance. Should any traces of nitric acid be perceived in an experiment of this kind, an unattacked portion of cloth or stuff must be examined, before we can draw the inference that nitric acid has been spilled or thrown on it. On these occasions we may be often disappointed in searching for chemical evidence of nitric acid. Not to mention that the acid may be easily removed by washing while the stain remains, we must remember that it is volatile, easily decomposed, and its nature entirely changed by contact with the organic substance. These facts will explain to us, why after a few weeks the chemical evidence of the presence of this acid will sometimes be entirely lost; while in the case of sulphuric acid, the stains may furnish abundant evidence of its presence after many years' exposure. In all cases of the suspected throwing of nitric acid, the spots on the dress should be examined as soon as possible, or the chemical analysis will fail. The following case occurred a few years since at Guy's Hospital:—A man had some strong nitric acid maliciously thrown in his face, and the sight of one eye was thereby entirely destroyed. He wore at the time a blue stuff coat, which was not sent to be examined until *five weeks* after the accident, and only a few days before the trial of the prisoner for the offence! The sleeve and body of the coat were found to be covered with numerous spots of a yellowish-brown colour. The spots were quite dry; they had evidently been caused by some corrosive acid. The colour was discharged, and the fibre of the stuff corroded. Not a trace of nitric acid could be detected in them, although there was no doubt that it had been used. Its disappearance was probably due partly to its decomposition in the stuff, and partly to its volatility. Had the coat been examined soon after the offence, the nature of the acid would have been easily determined. I have been able to procure certain evidence of the presence of nitric acid in stains on black cloth, a fortnight after the liquid had been spilled. The quantity of acid present was, however, so small, that on adding to the filtered liquid, gold leaf and muriatic acid, and boiling, there was no apparent solution of the metal, nor on trying another portion with sulphate of indigo was the colour discharged. A third portion of the acid liquid was neutralized by carbonate of potash, and evaporated, when crystals of nitre (amounting to about a grain) were obtained. These rapidly gave, with copper filings and sulphuric acid, the characters of a nitrate. There was but a thin slip of cloth used in the experiment. Dr. Christison has obtained evidence of the presence of the acid in stains on cloth, made seven weeks before. (Op. cit. 178.) The neutralized liquid in such cases will often be found to give a well-marked effect with sulphate of narcotine. In conducting an analysis of this kind, it has been recommended, when we obtain an acid liquid, to test it with the nitrate of barytes and nitrate of silver. The liquid, if it contain nitric acid only, should give negative results; but there are few specimens of cloth which do not yield traces of sulphuric and muriatic acids or of sulphates and muriates or chlorides, so that nitric acid may still be present when one or the other of these tests is affected.

QUANTITATIVE ANALYSIS.

Convert the nitric acid, contained in a measured quantity of the liquid, to nitre, in the way above described. Convert the whole quantity of nitre thus obtained to sulphate of potash by the cautious addition of strong sulphuric acid. Dissolve this salt in water, and evaporate to dryness. Calcine the residue, and afterwards wash it with alcohol, to remove if necessary any portion of free sulphuric acid. For every one hundred grains of dry sulphate of potash thus obtained, we may estimate that there were present in the measured quantity of liquid, about eighty-two grains of liquid nitric acid, (aqua-fortis, bihydrate)—the bulk of which by measure, may be easily determined by calculation. Should the dried sulphate be very acid it may be necessary to moisten it with a solution of sesquicarbonate of ammonia, and re-expose it to heat, to drive off the volatile alkali with the surplus sulphuric acid. Sulphate of potash, it must be remembered, is a perfectly neutral salt.

CHAPTER XVII.

POISONING BY MURIATIC ACID OR SPIRIT OF SALT—RARELY TAKEN AS A POISON. SYMPTOMS—POST-MORTEM APPEARANCES—QUANTITY REQUIRED TO DESTROY LIFE—FATAL CASES—TREATMENT. CHEMICAL ANALYSIS—DETECTION OF THE ACID IN PURE AND MIXED LIQUIDS—ON ARTICLES OF CLOTHING—IN CASES OF FORGERY. PHOSPHORIC ACID—EXPERIMENTS ON ANIMALS—A COMPARATIVELY INERT SUBSTANCE—TESTS FOR PHOSPHORIC AND PYROPHOSPHORIC ACIDS. BORACIC ACID.

But very little is known concerning the action of muriatic acid or hydrochloric acid as a poison. That this form of poisoning is not very common, may be inferred from the fact, that Orfila has reported only one case, communicated to him by Dr. Serres, in his large work on Toxicology (i. 155.) In the Coroner's report for England, during the years 1837–8, out of five hundred and twenty-seven cases of poisoning, there was not one in which this acid was the poison used. I have been able to collect the reports of only *three* cases of poisoning by muriatic acid that have occurred in this metropolis, during a period of sixteen years. From this statement it will be seen that the medico-legal history of this kind of poisoning is very incomplete.

SYMPTOMS.

From the few imperfect reports that have yet appeared, the symptoms produced by this acid do not differ widely from those caused by the two other mineral acids, described in the preceding chapters. There is the same sensation of burning heat extending from the throat to the epigastrium, with vomiting of a highly acid liquid of a dark green colour, mixed with mucous and altered blood. The tongue becomes swollen and dry; and with much thirst, there is great difficulty of deglutition. The tonsils and throat are inflamed. It is said that there is an escape of acrid pungent vapours from the mouth, when the acid is first swallowed (Orfila;) but this does not seem to have been observed in any of the reported English cases. In two instances, neither the vomiting nor pain in the abdomen was urgent, although both terminated fatally. The chief seat of pain was in the throat and fauces. In one instance, in which probably an ounce of the acid had been swallowed, the individual was able to walk to his home at a distance of three quarters of a mile. In general, the power of locomotion appears to be destroyed in mineral-acid poisoning, from the extreme severity of the pain (p. 164, ante.) The pulse has been observed to be small, frequent, and irregular; the skin cold and clammy. The intellectual faculties have remained clear until death.

POST-MORTEM APPEARANCES.

The fauces, larynx, and œsophagus, have been found highly inflamed, the mucous membrane lying in detached masses or actually sloughing away. In one instance the membrane was thickened. The coats of the stomach have been so much corroded that, in many places, there was only the peritoneal tunic left; and in attempting to remove the organ in this case, the parietes gave way. The contents have been sometimes of a yellowish, at others of a dark green colour. In one case, where the fundus of the gall-bladder came in contact with the stomach, it was observed to have a bright green colour, arising from the well-known action of this acid on the bile. In no instance yet reported, was the stomach perforated. On removing the contents, the lining membrane has been found blackened, and presenting a charred appearance—the blackening extending through the whole length of the duodenum, and being especially marked on the prominent parts of the numerous valvulæ conniventes, the intervals being stained of a greenish-yellow colour, from the action of the acid on the bile.—(Case by Mr. Quekett, Med. Gaz. xxv. 285.) When death did not take place until after the lapse of several days, the coats of the stomach were highly inflamed, and for the most part in a sloughing state;—large dark shreds of membrane were hanging from the sides of the organ, especially about the pylorus. The inflammation had extended also into the duodenum.

In a case which occurred in 1839, related by Dr. Galtier, a man swallowed hydrochloric acid (quantity not known) and suffered immediately from the usual symptoms. Magnesia and other remedies were employed, but he died in twelve hours. On inspection, nine hours after death,—the gall-bladder was found distended, and of a bright yellow colour, the stomach of a leaden colour, and its vessels gorged with black blood,—the coats so much destroyed that in some parts only the peritoneum remained, and they gave way on being handled. The mucous membrane was unequally blackened and corroded in patches. The same appearance was presented by the mucous membrane of the duodenum, especially at the edges of the valvulæ conniventes. In the interspaces the membrane was stained with a greenish-yellow coloured bile. (Toxicologie, i. 216.) It does not appear that any attempt was made to determine the presence of muriatic acid in the contents of the viscera: the case is thus left somewhat incomplete.

QUANTITY REQUIRED TO DESTROY LIFE.

With respect to this question, and *the period* at which the case proves fatal, there is no reason to suppose that the muriatic acid differs from the sulphuric and nitric acids in relation to these points. The cases that have hitherto occurred throw but little light upon these questions. The medical jurist must be content to draw an inference, the fairness of which cannot be disputed, when it is based upon the strong analogy which exists between the effects of this and the other two acids. The facts at present before us are these.—In one case, two ounces destroyed life in thirty-three hours; in a second, the same quantity killed the person in eight days; and in a third, a like dose proved fatal *in five hours and a half*. This, I believe, is the most rapidly fatal case on record. The smallest dose that appears to have destroyed life was about an *ounce*. The patient died in fifteen hours. In the case reported by Orfila, the dose was an ounce and a half, and this proved fatal in about eighteen hours. If cases of poisoning by this substance were more frequent, it would, no doubt, be found that not only might death take place within a much shorter period, but that a much smaller quantity, even one drachm, might prove fatal. All the cases of poisoning by this acid, which I have found reported, have occurred in adults; some from accident, and others from suicide. There is no instance, so far as I know, in which muriatic acid has ever been used for the purpose of murder.

[Dr. Toothaker (Boston Med. and Surg. Journ. xv. 270,) reports a case where an ounce of the officinal acid was taken, and the patient recovered, though with long continued symptoms of irritation of the gastric mucous membrane.—G.]

The following case reported by Mr. Crawford, (Lancet, March 1840,) is in several respects interesting to the medical jurist.

A woman aged forty, took, in order to poison herself, two ounces of a mixture used by her husband for the purpose of browning gun-barrels. This mixture was stated to be composed of equal parts of tincture of steel and strong muriatic acid, with a few drops of a solution of corrosive sublimate. Soon after she had taken the liquid, she vomited. In about half an hour, she was seen by a druggist; vomiting had then ceased. She answered questions rationally, and was sensible until she died; but made no complaint of heat or pain in the mouth, throat, or epigastrium. There was no thirst. The pulse could not be felt at the extremities, and the heart's action was very feeble. The muscles of the extremities felt hard, as if contracted. She died in about five hours and a half after taking the mixture. Two hours before death, the bowels were opened twice. On inspection, the stomach was found contracted, and its mucous membrane thrown into ridges and furrows. The ridges were of a brown colour, as if charred; when the surface was scraped, it could be readily peeled off, and the part exposed showed numerous small black granules, which seemed to be nothing more than altered blood. The furrows were of a fine scarlet colour. It was not perforated. Appearances similar to these were noticed in the duodenum and jejunum. The lower part of the œsophagus was charred, and its lining membrane was easily peeled off. There is no account given of the state of the mouth, fauces and larynx. There is no doubt that the active agent and cause of death here was muriatic acid. An analysis of the mixture proved this, for it yielded an abundance of that acid, and a quantity of iron; but there was no trace of bichloride of mercury.

TREATMENT.

The same as in poisoning by sulphuric and nitric acids. (See ante, p. 169.)

CHEMICAL ANALYSIS.

The commercial acid has a deep lemon-yellow colour. It contains iron, and often common salt, leaving a residue of impurity on evaporation. It is not commonly so *concentrated*, as to possess the property of fuming in the air: a property which of course depends on its strength, and therefore may be present or absent in any given specimen. The liquid will be found highly acid:—it tinges organic substances of a yellow colour, and corrodes them. 1. When boiled with copper, there is but little action, the acid is in great part distilled over in vapour. 2. The acid, if moderately pure, may be boiled entirely away on pure mercury without being affected by the metal. These tests eminently distinguish the muriatic from the two preceding acids. 3. When boiled with black oxide of manganese, in fine powder, chlorine is evolved, known by its colour, odour, and bleaching properties on litmus and other colouring matters. This last test is conclusive:—there is no other acid which is thus affected by the peroxide of manganese. One drop of muriatic acid in a tube of very small bore, will give satisfactory results.

Tests.—When the acid is much *diluted* with water, the property of evolving chlorine with peroxide of manganese, is lost. In this case, there is one most satisfactory test for the presence of muriatic acid—the *nitrate of silver*. This test gives, with the acid, a dense white clotted precipitate of chloride of silver. The precipitate thus formed acquires speedily a dark colour by exposure to light; and it is known from all other white salts of silver, by the following properties. 1. It is insoluble in nitric acid. 2. It is very soluble in caustic ammonia. 3. It is insoluble in caustic potash. 4. When dried, and heated on platina or glass, it melts like a resin, forming a yellowish-coloured sectile mass. Unless these properties be possessed by the precipitate, it is impossible to refer the action of the test to the presence of muriatic acid. The delicacy of the silver test is such, that it will easily detect the thirteen-hundredth part of a grain of muriatic acid in a minimum of water, and will form a perceptible opalescence (precipitate,) when the acid is diffused through 290,400 times its weight of water.

Objections.—It may be objected to the application of the silver test, that other acids form with it white precipitates, which might be mistaken for the chloride of silver. There are two common acids, both of them poisons,—namely, the prussic and the oxalic, which in this respect, resemble the muriatic. The prussic acid would be immediately known by its odour, or by the effect of heat on the cyanide of silver. (See Prussic Acid.) The white precipitate produced by the test in oxalic acid, is known from the chloride by its entire solubility in nitric acid. The evaporation of a portion of the tested acid liquid, would moreover leave crystals of a solid acid. Iodic acid also gives a dense white precipitate with nitrate of silver, and the iodate of silver thus formed, resembles the chloride in being scarcely soluble in nitric acid (except when boiled in a large quantity,) and soluble in caustic ammonia. The iodic acid is a substance which is rarely met with out of a chemical laboratory. It is immediately known from the muriatic acid, by adding to the liquid, sulphurous acid and starch, when blue iodide of farina is produced. The iodate of silver differs from the chloride, in not being so readily changed by exposure to light: and in being decomposed by caustic potash, which separates from it brown oxide of silver. The oxalate of silver is also thus known from the chloride: oxide of silver, being separated from it by an alkali. The chloride is unchanged by caustic potash until heat is applied:—oxide of silver is then slowly produced. Caustic potash may therefore serve to distinguish these precipitates of the salts of silver from each other.

Again, the mixture of any simple acid, such as the acetic, tartaric, or citric, with a solution of common salt, might be pronounced to be muriatic acid from the action of the test, when in reality no free muriatic acid was present. A suspicion of this kind would naturally arise, if on evaporating a portion of the acid liquid, a large quantity of a solid white residue was obtained. The difficulty in such a case may be removed by resorting to the process recommended in speaking of sulphuric acid (ante, p. 176.) If we take equal quantities of the acid liquid, and precipitate one portion entirely by nitrate of silver,—then evaporate the other portion to dryness, dissolve the dry salt in water, and precipitate this solution entirely by the test, it is obvious that if there be no free muriatic acid present, the precipitated chloride will have exactly the same weight in the two cases. The precipitate should in each case be well washed in water, acidulated with nitric acid. If free muriatic acid were present, the precipitate obtained in the former case, would exceed in weight that obtained in the latter.

In liquids containing organic matters.—Such liquids will have a highly acid reaction. It might be supposed that the nitrate of silver would serve as a good trial-test; but it must be remembered, that this salt is liable to be precipitated by numerous organic liquids, such as vinegar and porter, although no free muriatic acid may be present. This arises either from the presence of chlorides in most liquids of this description, or from oxide of silver being itself precipitated by certain organic principles. In the last-mentioned case, the precipitate is known from the chloride by being entirely soluble in nitric acid. This test for muriatic acid cannot however be safely employed in the analysis of any liquid containing organic matter. Under these circumstances there are two ways of proceeding. 1. To distil the liquid at a low temperature in a retort fitted with a receiver. Any free muriatic acid will pass over, be condensed, and may now be safely tested. A mixture of sulphuric acid with a muriate in the liquid, would produce the same effect, and lead to error. The action of nitrate of barytes upon the acid organic liquid, would, however, show whether sulphuric acid were present or not. This process only answers when the muriatic acid is in moderately large proportion. If the quantity be small, none is obtained unless the distillation is carried to dryness; but then the process is open to objection. (See Ann. d'Hyg., Oct. 1842, ii. 339.) 2. We may evaporate to dryness, a fractional portion of the organic liquid; calcine any residue, and observe whether on disgestion in water and filtration, we obtain a solution of a chloride; if not, we must neutralize the acid organic liquid by adding carbonate of soda—then evaporate and incinerate the residue. In this way, we obtain all the muriatic acid contained in the liquid as chloride of sodium. Should any chloride result from the first evaporation of the acid liquid, the quantity of muriatic acid thus obtained, must be deducted from that which results in the last-mentioned process.

[The nitrate of silver though a delicate test for muriatic acid, acts in a similar manner with the soluble muriates, and hence the formation of the characteristic precipitate can only indicate that muriatic acid was present, but does not demonstrate whether it was in a free state. Another difficulty arises from the fact of this acid always existing as a constituent of the gastric juice, of which it forms about 1-1500th, and in some morbid conditions of the stomach a much larger proportion. Hence, its presence in the stomach in minute quantity is not to be absolutely attributed to its ingestion, or that it was even remotely the cause of death.—G.]

Vomited matters and contents of the stomach.—The process is the same in the two cases. The liquid should be separated from the solid portions by filtration through cotton or paper. If acid, we must proceed as directed in speaking of the analysis of an organic liquid. In giving evidence on this point, a witness may be fairly asked, whether the natural secretions of the stomach do not owe their acidity to the presence of free muriatic acid. The experiments of Dr. Prout have proved that this is really the case:—that the gastric secretions are acid, owing to the presence of free muriatic and acetic acids. An objection of this kind is answered by the facts,—that the quantity of free muriatic acid, naturally contained in the gastric secretions, does not exceed the 1500th part by weight, *i. e.* it amounts to about five grains in sixteen ounces of liquid. (Prout.) This would give only a very feeble acidity, and but a trivial result with the test; whereas, the liquid may be intensely acid, and yield a large quantity of muriatic acid on being distilled. 2. The medical jurist would look for the characteristic symptoms and post-mortem appearances, before he inferred that the mineral acid had been taken as a poison. If these are wanting, and the quantity of free muriatic acid is but small, then there would be no evidence of poisoning, so far as chemical analysis was concerned. A mixture of vinegar and salt might be easily mistaken for muriatic acid in an organic liquid, but no muriatic acid would be obtained on distillation, and no additional quantity of chloride would be obtained on neutralization by carbonate of soda and incineration. As organic matter holds muriatic acid with strong affinity, Orfila has recommended that it should be precipitated by a strong solution of tannic acid, before distillation is resorted to. The liquid for examination may be *neutral*, owing to the administration of antidotes. The muriatic acid may have been neutralized by carbonate of soda or magnesia. This would be discovered on evaporation, and the quantity of resulting alkaline chloride would indicate the quantity of muriatic acid. But to any inference of this kind, there are very strong objections. If the quantity of chloride of sodium be *small*, the results might be referred to that portion of the salt which always exists naturally in the gastric secretions; if *large*, the chloride of sodium is so common an ingredient in most kinds of food, that its presence in the contents of the stomach might cæteris paribus be fairly ascribed to this source. On the whole, then, it is clear that the chemical evidence in poisoning by muriatic acid must fail, unless the acid be discovered in large quantity and in a free state, in the contents of the stomach; or unless there be at the same time corroborative evidence of poisoning from symptoms and post-mortem appearances. It need hardly be observed, that owing to violent vomiting or medical treatment, all traces of the acid may have disappeared from the stomach, notwithstanding the person may have died from its effects. It does not appear that muriatic acid was found in the stomach in the few cases of poisoning by it, which are on record. The analyst must remember, that in examining the stomach of a person poisoned by muriatic acid, he may discover arsenic,—some of the common varieties of the acid containing this poison as an impurity.

On solid organic substances.—Chemical evidence may be obtained from this source, when other sources fail. In Mr. Quekett's case, no muriatic acid was found in the stomach: but the nature of the poison was accurately determined by examining a piece of the deceased's waistcoat, on which some of the acid swallowed, had become accidentally spilled. By digesting the stuff in warm distilled water, a highly acid liquid may be obtained on filtration, which, if muriatic acid be present, will yield, with nitrate of silver, a white precipitate, possessing all the properties of chloride of silver. The spots produced on black in a platina capsule, and add finely powdered muriate of ammonia, at the same time applying a high temperature by a spirit lamp. If phosphoric acid be present, it will escape in dense white flakes as a volatile and strongly acid vapour (pyrophosphoric acid) plainly distinguishable from the white vapour of muriate of ammonia.

When phosphoric acid has been heated to a high temperature, it becomes converted to PYROPHOSPHORIC ACID, and its chemical properties are entirely changed. In this state, it may be obtained by acting on phosphorus with nitric acid, and evaporating to a syrup. When this syrupy extract or the solid acid is dissolved in water, it forms a strongly acid solution—which is precipitated *white* by nitrate of silver, whether an alkali be previously added or not. The pyrophosphate of silver is easily soluble in nitric acid. Pyrophosphoric acid also precipitates immediately a solution of albumen in water. A solution of common phosphoric acid does not precipitate this animal principle.

Phosphorous acid.—This acid differs from the phosphoric, in possessing the property of reducing at a boiling temperature the nitrate of silver, chloride of gold, and corrosive sublimate. The best method of analysing it is, to convert it by nitric acid and heat to phosphoric acid, and then apply the tests for this body. It should be remembered that the acids of phosphorous sometimes contain arsenic under the form of arsenic acid. The presence of a minute trace of arsenic may be immediately detected by the application of Reinsch's process. (See post, ARSENIC.)

BORACIC ACID.

According to Mitscherlich, this solid mineral acid is a very virulent poison, but it does not begin to act until some time after it has been administered to an animal. He found that one drachm killed a small rabbit in seventeen hours; half a drachm, given in two doses at an interval of less than four hours, caused the death of another rabbit in about twenty-six hours. The symptoms produced were rapid breathing and quick pulse, followed by debility. Spasms supervened, with colic (?), great restlessness, soft and mucous fæces, great prostration, and convulsions. Before death the breathing became slow and the body cold. The stomach and small intestines were found highly inflamed. He could not ascertain that the acid was absorbed; but Wöhler is reported to have found it in the urine of a dog to which he had administered it. (Med. Times, Sept. 1845, p. 342.) So far as I am aware, this substance has never produced any serious effects on the human body.

CHEMICAL ANALYSIS.

Boracic acid is seen in white scaly crystals. 1. It is not very soluble in water, but gives, when boiled in water, a well-marked acid reaction. 2. It is soluble in alcohol, and when the flame is ignited, this acquires a rich green colour. 3. It is soluble in caustic alkalies, forming the class of borates. If taken as a poison, it is likely from its great insolubility, to be found in the contents of the stomach, or in the matter ejected by vomiting.

CHAPTER XVIII.

POISONING BY MIXED MINERAL ACIDS—AQUA REGIA, OR NITRO-MURIATIC ACID—AQUA REGINÆ, OR NITRO-SULPHURIC ACID. TESTS FOR THE COMPOUND ACIDS—FEIGNED POISONING BY THE MINERAL ACIDS—DIAGNOSIS FROM SYMPTOMS—DIAGNOSIS FROM POST-MORTEM APPEARANCES—CASE—ACTION OF THE MINERAL ACIDS ON DEAD ANIMAL MATTER—DEATH FROM LATENT NATURAL CAUSES IN POISONING BY THE MINERAL ACIDS.

In general the mineral acids are taken separately as poisons; but they may be taken in a mixed state; especially as some mixtures of this description are largely used in the arts. Thus, the AQUA REGIA, a mixture of nitric and muriatic acids, is used for dissolving gold and platina; while the AQUA REGINÆ, nitro-sulphuric acid, is employed for dissolving silver and separating it from plated articles. I have not been able to find in any work on toxicology, an account of a case of poisoning by the nitro-muriatic acid; but Orfila gives one case of poisoning by nitro-sulphuric acid.

A man, aged twenty-four, swallowed a mixture consisting of one ounce of strong nitric acid and two drachms of strong sulphuric acid. The usual symptoms followed, and he died in eight hours. The post-mortem appearances, as might have been presumed from the relative quantities of the two acids taken, were more those of nitric than of sulphuric acid. (Toxicologie Générale, i. 129.) There is but little doubt that nitro-muriatic acid would produce symptoms, and cause appearances, analogous to those described in speaking of muriatic acid. The mixed effects of nitric acid might be also perceptible. The quantity required to destroy life, and the period at which death will ensue, may be inferred from what has been already said of these acids.

CHEMICAL ANALYSIS.

Nitro-muriatic acid.—This liquid is of a deep yellow or red colour, and is intensely acid.—1. It is known from its two component acids, by immediately dissolving pure leaf-gold, either with or without the aid of a gentle heat.—2. On boiling a portion of the mixture, chlorine gas is evolved; and litmus paper is bleached at the mouth of the vessel. If the mixed acids be very much diluted with water, these properties are lost.—3. Muriatic acid is discovered, by adding to it, when diluted, nitrate of silver.—4. Nitric acid is detected by boiling in it some cuttings of copper—the red fumes of nitrous acid are evolved. Unless there be sulphuric acid present, nitrate of barytes will give no precipitate with the diluted acid. It is difficult to understand, that it would ever be necessary for legal purposes, to determine the relative proportions of nitric and muriatic acids in this compound. If such a necessity arose, oxide of silver might be used to separate the muriatic acid, and the quantity of this deducted from the whole quantity of acid experimented on, would give the proportion of nitric acid. There are other methods of a purely chemical nature, for which chemical works may be consulted.

Nitro-sulphuric acid.—This acid may be easily mistaken for sulphuric acid, since it gives a white precipitate with a salt of barytes, and the nitric acid remains concealed.

Process.—Add carbonate of barytes in fine powder to the mixed acids, largely diluted, until they are neutralized. The sulphuric acid alone is precipitated, as sulphate of barytes. This may be separated by filtration, washed with water acidulated with nitric acid, dried and weighed, by which the quantity of sulphuric acid will be determined. The filtered liquid, on being evaporated, will yield crystals of nitrate of barytes, in which the presence of nitric acid may be easily determined by the usual tests. In the arts, it is common to employ a mixture of sulphuric acid and nitrate of potash, instead of nitric acid. In this case, the carbonate of barytes would precipitate the sulphuric acid, and leave the nitrate unaffected, so that the analysis is rendered more easy. Both the nitro-muriatic and nitro-sulphuric acids discharge the colour of sulphate of indigo at a boiling temperature.

In concluding this account of the mineral acids, it will be proper to consider the medico-legal questions that are likely to arise respecting this form of poisoning, in addition to those which have already been examined. There are some questions common to all the acids; and these were succinctly and clearly stated in respect to nitric acid by Tartra, in his Traité de l'Empoisonnement, published in 1802.

1. *Can poisoning by the mineral acids be feigned?*

It might seem superfluous to raise such a question, yet the author to whom I have above referred, relates an instance where a young lady pretended that she had swallowed aqua fortis, but the imposition was easily detected. Vomiting and pain in the abdomen may easily be feigned; but the fact is, there are too many well-marked peculiarities in poisoning by either of the three acids, more especially in relation to their local effects on the mouth and fauces, to render such an imposture successful.

2. *Can a medical jurist pronounce judicially from symptoms only, that a person has been poisoned by one or other of the mineral acids?*

If we find that the particular symptoms described, of intense burning pain in the throat and fauces, have immediately followed the swallowing of some sour liquid;—if the matter vomited, be highly acid,—dark-coloured and mixed with shreds of mucus and altered blood, there can be but little doubt, that the symptoms must have been caused by one of these mineral acids. At any rate, there is no disease, commencing suddenly in a healthy person, which would be likely to produce them. A doubt may arise, whether they may not have been caused by some other poison. An examination of the mouth and fauces will, however, assist the judgment. If a mineral acid were the cause, and it had been taken either concentrated or but slightly diluted, the lining membrane will be found corroded, discoloured, and dissolved;—in poisoning by sulphuric acid, of a brownish white colour, if recent, and by nitric acid, of a yellow or citron colour. With respect to this criterion, however, it must be remembered that the mucous membrane of the mouth has been found white in poisoning by each of these acids. Then, again, spots indicative of the nature of the acid may be found spilled on the skin or clothes. Whenever the cause of the symptoms is at all doubtful, no opinion should be expressed in favour of poisoning, unless the mineral acid be actually discovered in the matters vomited from the stomach.

3. *Can a medical jurist pronounce from post-mortem appearances only, whether the deceased has been poisoned by one or other of the mineral acids?* —Zacchias, Plenck, Morgagni, and many medico-legal writers, have laid it down as an axiom in legal medicine, that the only certain evidence of poisoning, is the discovery of the poison itself in the stomach or intestines. According to Plenck; "Unicum signum *certum* dati veneni est *notitia botanica* inventi veneni vegetabilis, et *criterium chemicum*, dati veneni mineralis." It is fortunate that the law does not act upon this doctrine; for as there are very good medical reasons, why a poison, when actually the cause of death, should not always be found in the stomach, it is clear, if this proof were required in all cases, that a conviction for poisoning would frequently rest upon pure acci-

dent. Perhaps there is no form of acute poisoning, in which a medical jurist has it more in his power to pronounce an opinion from post-mortem appearances, than in these cases of death from the mineral acids. Tartra long since observed, that whenever the alimentary canal from the mouth to the intestines, was found corroded and converted to a soft fatty substance of a bright yellow or brown colour, when it was easily detached from the subjacent parts, and there were marks of inflammation or gangrene or actual perforation of the stomach, there could be no doubt that these effects were due to a mineral acid, whether the acid were discovered in the body or not. Dr. Christison has also adopted this view; and he very properly remarks that such cases must be considered as distinct exceptions to the general rule, regarding the weakness of evidence derived from post-mortem appearances. Indeed, it may be inquired of those who are disposed to entertain an adverse opinion, what conceivable form of disease can produce such well-marked appearances simultaneously in the mouth, fauces, œsophagus, stomach and intestines, in the course of a few hours? In the case of *Humphreys*, given in a former chapter (p. 47, ante,) the prisoner was properly convicted of murder by poisoning with sulphuric acid, although no trace whatever of that poison could be discovered in the body. On these occasions, it would not be safe to trust to appearances in the stomach alone. I have seen the mucous membrane of this organ very much blackened, although *not corroded*, in a case where death had taken place from natural causes, *i. e.* perforation of the stomach. The appearance was precisely like that which was met with in a case of poisoning by sulphuric acid (p. 105, ante.) It is therefore necessary, in every suspected case, that attention should be paid to the condition of the mouth, fauces, and œsophagus. The spontaneous and morbid changes to which the stomach is liable, do not affect these parts. If the latter be healthy and unaffected, we cannot safely infer, in a doubtful instance, whatever may be the condition of the stomach, that a strong mineral acid has been taken.

This question respecting the value of evidence from post-mortem appearances in alleged poisoning by sulphuric acid, was raised in the case of *The Queen* v. *Thomas*. (Monmouth Lent Assizes, 1847.) There were some remarkable points about this case (ante, page 167.) The deceased was an infant ten days old,—the acid which was stated to have caused its death was diluted with twenty-nine parts of water. There were the usual symptoms, and the child died in two days. No sulphuric acid was found in the stomach, and the medical evidence of the cause of death was chiefly based on the post-mortem appearances. According to the medical witness who examined the body, the mouth was in a very *healthy* condition, but in the back part of the throat, the mucous membrane was *corroded* and detached in several places:—the same was the case down to the stomach. The stomach was slightly inflamed in patches: the mucous membrane was so much softened that it could be easily removed by being scraped with the nail: the small intestines were also *corroded*, but in a less degree than the stomach. Another medical witness deposed that there was corrosion, amounting to almost total destruction of the lining membrane of the œsophagus, stomach, and intestines. The mouth, with the exception of the lower lip, presented no sign of excoriation or abrasion. (Pharmaceutical Times, April 24, 1847, 162.)

The defence rested upon two assumptions:—1. That the spot on the lip might have arisen from a diseased nipple in the nurse. 2. That as there were no traces of the action of the acid in the mouth, the appearances in the œsophagus, stomach, and intestines, might have been produced by *natural* causes! The learned judge who tried the case leaned to this view in charging the jury, and the prisoner was acquitted.

The simple medical question in this case is—Were the witnesses correct in stating that there was *corrosion* of the œsophagus, stomach, and intestines? The view adopted by them was supported by the fact that the child had been seen to vomit black and red matter, and its clothes were found to be corroded in various places by the action of some acid. The non-discovery of sulphuric acid in the body does not at all conflict with their opinion that death had been caused by it, and they very properly declared that no *natural cause*, operating within the short period of two days, could have produced the appearances found in the alimentary canal. There is no reason to disbelieve this evidence, and the absence of marks or injury to the mouth might have been probably due to the acid having been poured down the throat gradually by a spoon. This would be rendered all the more easy by its being considerably diluted with water. It is certain, according to the medical evidence, that no natural disease could have produced corrosion of the œsophagus, stomach and intestines: and the prisoner therefore owed her acquittal to a total misapprehension of the medical facts. It need hardly be said that the sore nipple of a female cannot lead to the chemical destruction of the alimentary canal of a child, although it may slowly affect the lips! Such a case as this reflects but little credit upon the mode in which criminal prosecutions are carried on in this country. It would seem as if a desire occasionally existed to make complex and unintelligible to the minds of a provincial jury, medical facts which, if clearly observed, can only admit of one construction.

In cases of *chronic* poisoning, the appearances in the body may be obscure, and present nothing characteristic.

4. *A mineral acid may be introduced into the dead body. Is it possible to determine whether the corrosive poison has been taken during life, or designedly introduced after death?*—This question is purely speculative; for it is difficult to understand, how a strong mineral acid should be introduced into the stomach of a dead subject, even supposing that any object for such an act should exist. The only motive would be the imputing of a charge of poisoning to another, which might be disproved by the history of the case, without reference to the discovery of poison in the dead body. In order to distinguish such a case the following points may be attended to. Those who die from this kind of poisoning, generally live sufficiently long to allow of the establishment of well-marked vital reaction in the organs affected. There may be not merely a chemical corrosion of parts, but marks of inflammation, gangrene and the effusion of lymph. If the poison were swallowed during life, these effects would be apparent from the mouth and fauces downwards. The sulphuric and nitric acids will stain and corrode the parts of a dead body with which they are in immediate contact; but they will not produce any appearance of vital reaction in the surrounding structures. I have not found that strong sulphuric acid readily charred the dead stomach or duodenum until its effect was aided by heat, nor did it ever produce a red or inflamed appearance of the mucous membrane.

5. *A person has taken a mineral acid and died. Is it possible to determine whether death was produced by the acid or by some other latent cause?*—This question has already been fully discussed in relation to general poisoning (p. 143, ante;) but it is much more simple and more easily answered, when it is restricted to poisoning by the mineral acids. The symptoms preceding death from these poisons, and the well-marked chemical effects produced by them from the mouth downwards, will readily enable a medical jurist to form an opinion of the real cause of death. The symptoms produced by perforation of the stomach from disease, somewhat resemble this kind of poisoning; but the course of this disease is to destroy life by inducing peritonitis. It would be, indeed, a singular coincidence, that perforation of the stomach from disease, and poisoning by a mineral acid, should occur simultaneously in a person: and in

such a case, a decision might be difficult. The inference would probably be that the perforation was caused by the poison. (See ante, p. 146.) Orfila mentions an instance where a patient in a public hospital, had an ounce and a half of muriatic acid given to him by mistake, and he died under the usual symptoms within twenty-four hours. On inspection, the lips were found black, the tongue brown, thickened, hard and dry:—the pharynx and œsophagus were of a deep purple red colour, and excoriated in several places. The stomach was inflamed and gangrenous—its mucous membrane separating in layers;—the duodenum was in the same state. The jejunum was perforated by a lumbricus which was found in the cavity of the abdomen. (Toxicologie, i. 155.) There was no doubt in this case, from the symptoms and post-mortem appearances, that death was immediately due to the action of the mineral acid. When the disease is merely of a chronic nature, there would be no difficulty in forming a diagnosis.

CHAPTER XIX.

POISONING BY THE VEGETABLE ACIDS. OXALIC ACID—SYMPTOMS AND EFFECTS—POST-MORTEM APPEARANCES—EXPERIMENTS ON ITS ALLEGED CORROSIVE ACTION ON THE STOMACH—PERFORATION—FATAL DOSES—RECOVERY FROM LARGE DOSES—PERIOD AT WHICH DEATH TAKES PLACE—TREATMENT—CHEMICAL ANALYSIS—TESTS FOR OXALIC ACID IN PURE AND MIXED LIQUIDS. OXALIC ACID IN ORGANIC SUBSTANCES—ITS ALLEGED PRODUCTION. POISONING BY THE RHUBARB PLANT. TARTARIC ACID—CHEMICAL ANALYSIS. CITRIC ACID. ACETIC ACID. VINEGAR.

OXALIC ACID.

Oxalic acid is one of the most powerful of the common poisons; but its use as a poison is almost entirely confined to this country. Cases of poisoning by it are generally the result of suicide or accident. In the Coroners' return for 1837–8, there were nineteen cases of poisoning by this substance; out of which number fourteen were the result of suicide. It is singular, also, that the greater number of these occurred in the county of Middlesex. Accidental poisoning by oxalic acid has arisen from its strong resemblance to Epsom salts. It is not often that we hear of its being used as a poison for the purposes of murder. Its intensely acid taste, which could not be easily concealed by admixture with any common article of food, would infallibly lead to detection, long before a fatal dose had been swallowed. I have known several trials to take place for attempted poisoning by oxalic acid,—in two the vehicle selected for its administration was coffee or tea, and in one which occurred recently, the poison was powdered and mixed up with brown sugar to conceal the taste. (*Reg.* v. *Dickman*, Central Criminal Court, February 1845.)

SYMPTOMS.

In many instances of poisoning by this substance, death has taken place so rapidly, that the individual has not been seen alive by a medical practitioner. If the poison is taken in a large dose, *i. e.* from half an ounce to an ounce of the crystals dissolved in water, a hot burning acid taste is experienced in the act of swallowing, and vomiting occurs either immediately or within a few minutes. Should the poison be diluted, there is merely a sensation of strong acidity, and vomiting only occurs after a quarter of an hour or twenty minutes. In some instances, there has been very little or no vomiting; while in others, this symptom has been incessant until death. In one case, where the poison was much diluted, vomiting did not occur for seven hours. (Christison, 221.) The vomited matters are highly acid, and have a greenish brown or almost black colour; they consist chiefly of mucus and altered blood. In one instance, reported by my friend, Dr. Geoghegan, they were colourless. (Med. Gaz. xxxvii. 792.) There is at the same time great pain and tenderness in the epigastrium, followed by cold clammy perspiration and convulsions. In a case which occurred at Guy's Hospital, in May 1842, where about two ounces of the poison had been swallowed, there was no pain. Urgent vomiting and collapse were the chief symptoms. There is in general an entire prostration of strength, so that if the individual be in the erect position, he falls; there is likewise unconsciousness of surrounding objects, and a kind of stupor, from which, however, the patient may be without difficulty roused. Owing to the severity of the pain, the legs are sometimes drawn up towards the abdomen. The pulse is small, irregular, and scarcely perceptible; there is a sensation of numbness in the extremities, and the respiration, shortly before death, becomes spasmodic. The inspirations are deep, and a long interval elapses between them. Such are the symptoms commonly observed in a rapidly fatal case.

Should the patient survive the first effects of the poison, the following symptoms appear: There is soreness of the mouth, constriction in the throat, with painful deglutition,—tenderness in the abdomen, with irritability of the stomach, so that there is frequent vomiting, accompanied by diarrhœa. The tongue becomes swollen, and there is great thirst. The patient may slowly recover from these symptoms. In a case related by Mr. Edwards to the Westminster Medical Society, the patient, a female, lost her voice for eight days; but whether this depended on the action of the oxalic acid which she had taken, or not, it is difficult to say. Certain it is, that this poison has a remote effect on the nervous system, indicated by the numbness and tingling in the extremities, which have been observed to remain long after the patient has recovered from the first effects.

From Dr. Christison's experiments, it would appear that this acid is still a poison, even when so diluted as to lose all its irritant and corrosive properties. It thus differs from the mineral acids. The effects vary according to the dose. In a large dose, but much diluted, the poison appears to destroy life by producing paralysis of the heart. When the dose is diminished, the spinal marrow is affected, and tetanus is one of the symptoms: when still less, but enough to prove fatal, the poison acts like a narcotic, and the animal dies as if poisoned by opium. (On Poisons, 219.)

POST-MORTEM APPEARANCES.

The lining membrane of the mouth, fauces, and œsophagus, is commonly white, although it is often coated with a portion of the dark brown mucous matter discharged from the stomach. This latter organ contains a dark-brown mucous liquid, often acid and having almost a gelatinous consistency. On removing the contents, the mucous membrane will be seen pale and softened, without always presenting any marks of inflammation or abrasion, if death have taken place rapidly. This membrane is soft and brittle, easily raised by the scalpel, and presents much the appearance, which we might suppose it would assume, after having been for some time boiled in water. The small vessels are seen ramifying over the surface, filled with dark-coloured blood, apparently solidified within them. The lining membrane of the œsophagus presents much the same characters. It is pale, and appears as if it had been boiled in water, or digested in alcohol; it has been found strongly raised in longitudinal rugæ, interrupted by patches where the membrane has become abraded. With respect to the intestines, the upper portion of the canal may be found inflamed; but unless the case be protracted, the appearances in these viscera are not very strongly marked. In a recent case of poisoning by this acid, where two ounces had been taken, and death was rapid, the coats of the stomach presented almost the carbonized appearance produced by sulphuric acid, owing to the colour of the altered blood spread over them.

Oxalic acid does not appear to have a strong corrosive action on the stomach, like that possessed by the mineral acids. It is therefore rare to hear of the coats of the organ being perforated by it. In many experiments on animals, and in some few observations on the human subject, I have found nothing to bear out the view that perforation is a common effect of the action of this poison. The acid undoubtedly renders the mucous coat soft and brittle, and it dissolves by long contact animal matter, which on analysis is found to be gelatin. Its solvent powers on the animal membranes are not, however very strong, as the following experiment will show. A portion of the jejunum of a young infant cut open, was suspended in a cold saturated solution of oxalic acid for six weeks. At the end of this time, the coats, which were white and opaque, were well preserved, and as firm as when they were first immersed, requiring some little force with a glass rod to break them down. On boiling a portion of the solution of oxalic acid, there was no change, nor did nitric acid produce any precipitate. On adding a weak solution of tannic acid (which is not affected by oxalic acid,) a precipitate of tanno-gelatin fell down. It was therefore obvious that a small quantity of the gelatin of the tissues had been dissolved, proving the independent existence of that principle in the animal membranes. From the slight solvent power here manifested on the thin coats of the intestines of an infant, I am inclined to doubt whether the contact of the acid with the adult stomach after death, would lead to perforation of the organ. A case reported by Dr. Geoghegan, which will be presently mentioned, is decidedly against such a view.

Dr. Christison refers to only one instance in which, after death from oxalic acid, the stomach was found perforated. Orfila and Galtier do not mention a single case. One instance, however, has lately fallen under the notice of Dr. Letheby. An unmarried female, æt. 22, of previously good health, swallowed one evening, in a fit of jealousy, a dose of oxalic acid (quantity not known,) and the next morning she was discovered dead in her room. On inspection, the stomach was found much corroded and softened. The mucous membrane was much blanched, except in two or three places, where there were small black spots, as if blood had been effused and acted upon by the poison; and here and there a blood-vessel might be seen ramifying, with its contents similarly blackened. The coats of the stomach were so softened, that it could scarcely be handled without lacerating it. At the cardiac end the coats were of a pulpy or gelatinous consistency, and presented numerous perforations. The contents amounted to six ounces, and were of a dark colour like porter, with but little solid matter. The liquid was strongly acid; and on being tested was found to contain about three drachms of oxalic acid. The softening effect here was probably due to long contact of the acid after death. (Med. Gaz. xxxv. 49.)

Dr. Geoghegan has recently published a detailed account of the appearances met with in the stomachs of three persons poisoned by oxalic acid. The first was taken from the body of a young man, who must have died in about *twenty minutes* after swallowing the poison. The inspection was made the following day. The stomach contained about eight ounces of a dark brown and viscid matter, resembling coffee-grounds, evidently largely impregnated with altered hæmatosine, and possessing an acid reaction. The mucous membrane of the splenic end and portion of the body was of a deep blackish-brown colour, and of variable intensity, which permitted in part the moderately coarse ramiform vascularity of the submucous texture to appear through it as a blackish arborescence. The discoloured condition of the membrane extended in narrow streaks into the body of the organ, where the lining membrane was otherwise of a uniform light purple red colour. Near the pylorus the membrane was translucent, and again exhibited the dark ramiform vascularity of the submucous coat. The mucous tunic of the cardiac portion was extremely soft and thin, detachable only as a pulp, and in parts eroded. In the body of the stomach the lining coat was somewhat thickened, but less soft, removable in flakes of one-third of an inch; at the pylorus not thickened, and yielding strips of one-fourth of an inch. The other organs were not examined except the heart, the blood in the right cavities of which was fluid; and the colon, the transverse arch of which was much contracted.

In reference to the three cases Dr. Geoghegan makes the following judicious observations. Although in one of them, the contents, including no inconsiderable amount of acid, remained in contact with the coats of the organ, no perforation was observable, the solvent energies being diffused over a large surface. The dose was not ascertained in any of the cases. Oxalic acid and gelatin were discovered readily in the contents in all. The quantity of poison in the contents (an inquiry constantly and ignorantly made by counsel) was in the first case, about three to four grains; a much larger amount in the two latter, especially in No. 3. A comparison of these cases with those already on record gives as the ordinary appearances in the stomachs of persons who have succumbed to the influence of oxalic acid—1. Contents, of the colour of coffee-grounds, consisting of altered insoluble hæmatosine and mucus, and separating into a supernatant fluid and insoluble deposit. 2. Softening of mucous membrane, with various shades of brown coloration, erosion, or gelatinization. 3. Brownish-black ramiform vascularity of submucous tissue, owing to the imbibition of the acid contents. It is particularly important to note in similar cases the coexistence of this latter condition with the state of the contents just described, as the ramiform vascularity, or even diffuse brown discoloration, may and does present itself in many instances as the result of the action of the lactic acid of the gastric juice only. It appears evident that the fatal result in cases of poisoning by oxalic acid cannot be referred to the corrosion of the stomach as its chief cause, but rather to the contemporaneous and energetic action which it exerts in arresting the circulatory forces. (Med. Gaz. xxxvii. 792.)

In protracted cases, the œsophagus, stomach, and intestines, have been found more or less inflamed. In some instances there have been scarcely any perceptible morbid appearances produced.

[Death may ensue from oxalic acid, and yet no traces of its action on the alimentary canal be observable on dissection; this occurred in a case where an ounce had been taken. (*Lond. Med. Repos. III.* 380.)—G.]

QUANTITY REQUIRED TO DESTROY LIFE.

A trial for murder by this poison took place in 1832, in which this question was put judicially to the medical witnesses. One deposed, that he thought ten grains of the acid was sufficient to destroy life, another that it was not sufficient. The prisoner was acquitted. A question of this kind can only be solved by a reference to recorded facts; but unfortunately, in most cases, it has been impossible to determine exactly the quantity of poison taken.

Oxalic acid, it is to be observed, presents some singular anomalies in its effects. In one case, a man swallowed, as nearly as could be ascertained, three drachms of the crystals:—there was immediate vomiting, but no other urgent symptoms, and he recovered in a few hours. In a second instance, a woman took nearly half an ounce of oxalic acid—the usual symptoms appeared

—she recovered in six days, and was able to leave the hospital. Mr. Semple met with a case, where a girl swallowed about *two drachms* of the poison dissolved in water. Vomiting occurred immediately. In about twelve hours the more urgent symptoms had disappeared; but there was still tenderness of the epigastrium, with irritability of the stomach. In the course of a few days the patient was quite well. In February 1842, a case occurred at King's College Hospital, where a girl had swallowed *two drachms* of the acid, dissolved in beer. The only symptom from which she suffered on admission, was pain. She entirely recovered the next day. Dr. Babington, of Coleraine, reports a case (Med. Gaz. xxvii. 870,) in which a girl swallowed by mistake two scruples (*forty grains*) of the poison. Severe symptoms followed, chiefly marked by great gastric irritation. It was a week before this girl was convalescent, and a much longer time elapsed before she was able to resume her duties. In all of these cases, it is to be observed, proper medical treatment was resorted to; and the effects of the poison may be therefore supposed to have been in a great degree counteracted. But this explanation is hardly sufficient to meet all cases, as the following instance will show. A girl, æt. 15, swallowed two pennyworth (half an ounce) of oxalic acid, and she was not admitted into St. Thomas's Hospital until half an hour afterwards: a period had therefore elapsed, within which death has frequently taken place. When admitted she complained of great heat, and a sense of burning about the throat and fauces, with a feeling of sickness at the scrobiculus cordis; and there was vomiting of a large quantity of bloody frothy mucus. The stomach-pump was used, and a quantity of prepared chalk in water was injected:—lime water would have been preferable. After this she appeared sinking; collapse was coming on; the blood left the surface; the extremities were cold, and the pulse was hardly perceptible. Stimulants were given, and artificial warmth applied. The next day there was great soreness of the mouth and tongue, and the latter was swollen, red, and tender; skin hot; tenderness on firm pressure of the stomach. In a few days she perfectly recovered. (Med. Gaz. i. 737.) In the summer of 1846 I saw a similar case, where a like quantity had been taken, in Guy's Hospital: and here the extremities were very cold; but there was little pain on pressure of the abdomen some hours after the poison had been taken. This female recovered. It is not improbable that idiosyncrasy may account for these anomalies: *i. e.* that certain constitutions are with difficulty affected by this poison.

A smaller dose than *half an ounce* of the crystals, has not, so far as I am aware, been known to prove fatal; although, from the serious effects which have followed doses less than this, it is probable that a smaller quantity might destroy life when medical treatment was not resorted to. Two cases have occurred at Guy's Hospital, where in each, half an ounce of oxalic acid had been swallowed. Active treatment was adopted, and both patients recovered. When the dose is upwards of half an ounce, death is commonly the result; but one of my pupils informed me of a case where a man recovered, after having taken one ounce of the crystallized oxalic acid; and Dr. Brush of Dublin has more recently communicated to the Lancet, a case in which recovery took place perfectly after a similar dose of the poison had been taken. The acid was in this instance taken by mistake for Epsom salts. One ounce was put into a tumbler, and boiling water was poured on it at night. About half-past four in the morning the patient, a man aged sixty, stirred up the liquid and swallowed the whole. Contrary to what has been hitherto observed, there was no *immediate* vomiting:—the man, having discovered his mistake, tried to excite it, and only partially succeeded after the lapse of ten minutes. Warm water was freely given to him, and he ejected from his stomach dark grumous blood mixed with mucus. The usual antidotal treatment was then resorted to, and the stomach-pump used. In two hours symptoms of collapse appeared. In about six hours the skin had regained its warmth; but there was no pain in the stomach or any part of the abdomen. The secondary symptoms were a burning sensation in the mouth and throat, great difficulty in swallowing, thirst, acid eructations, and drowsiness; and these symptoms continued for two or three days. Vomiting and irritability of the stomach continued until the sixth day, but from this time the recovery was rapid; and in about eighteen days all unfavourable symptoms had disappeared. (Lancet, July 11, 1846, 39.) This must be regarded not only as an anomalous case in respect to symptoms, but as a remarkable instance of recovery from a very large dose. The report is incomplete, in not stating whether the vomited matters underwent analysis, and whether oxalic acid was found in them.

In the same journal is reported another case of recovery after an *ounce* of the acid had been swallowed. The man, it is stated, was not seen until fourteen hours after he had taken the poison; and he had, in the meantime, travelled a distance of ten miles to Dublin. He had immediately taken warm water. On his arrival in Dublin, magnesia and rhubarb were given to him. He complained of a burning sensation in the pharynx and œsophagus: his tongue was coated: his pulse small, quick, and wiry. There was anxiety of countenance, with complete prostration of strength. The palate was vesicated, and the pharynx was highly inflamed: there was tenderness of the epigastrium, with vomiting of a dark grumous matter mixed with blood. The man ultimately recovered, but for a long time afterwards he complained of a sense of constriction in the œsophagus. (Lancet, Sept. 13, 1845, 293.) The reporter of this case states, that the quantity of the poison actually taken exceeded an ounce.

According to the experiments of Mitscherlich, two drachms of this poison killed a rabbit in a quarter of an hour, and half a drachm killed another in half an hour. Fifteen grains produced general disturbance of the functions, but did not prove fatal. It is strange that this experimentalist should assert that oxalic acid does not produce inflammation of the intestinal canal. There are several cases on record which prove that this is a mistake.

It may be proper to state, that this poison is retailed to the public at the rate of from a quarter to half an ounce for twopence, and one ounce for fourpence or sixpence.

PERIOD AT WHICH DEATH TAKES PLACE.

Equal quantities of this poison do not always destroy life within the same period of time. In two cases, in which about two ounces of the acid were respectively taken, one man died in twenty minutes,—the other in three-quarters of an hour. Dr. Christison mentions an instance, where an ounce killed a girl in thirty minutes; and another where the same quantity destroyed life in *ten minutes*. Dr. Ogilvy, of Coventry, has reported a case of poisoning by oxalic acid, in which it is probable that death took place within *three minutes* after the poison had been swallowed. The sister of the deceased had been absent from the room about that period, and on her return, found her dying. The *quantity* of poison taken could not be determined. The only other remarkable circumstance in the case was, that the coats of the stomach were so softened, that on an attempt being made to remove the organ, they were lacerated by the weight of the contents. The intestines and left lobe of the liver were also found softened, as if by transudation. This is the most rapidly fatal case on record. (Lancet, Aug. 23, 1845, 205; and Med. Gaz. xxxvi. 831.) Dr. Iliff has communicated to me the particulars of a case in which the wife of a druggist, who had taken a dose of oxalic acid, was found dead by the side of the counter within a few minutes after she had been seen living. The stomach contained a black viscid acid liquid. The mucous membrane was not destroyed, and there were no particular signs of inflammation. The veins were gorged with blood, which gave a very peculiar appearance. The tongue was extremely white, but neither the fauces, œsophagus, or alimentary canal, presented any marks of inflammation. The vessels of the brain were turgid, and the pupils were dilated.

When the dose of oxalic acid is half an ounce and upwards, death commonly takes place in an hour. There are, it must be admitted, numerous exceptions to this rapidity of action. Dr. Christison reports two cases, which did not prove fatal for thirteen hours; and in an instance that occurred to Mr. Fraser, in which only half an ounce was taken, the individual died from the secondary effects in a state of perfect exhaustion, twenty-three days after taking the poison.

[An interesting case was recorded by Dr. T. Jackson, (*Boston Med. and Surg. Journ.*, xxx. 17.,) where death did not occur until the tenth day.—G.]

TREATMENT.

It is recommended that water should be sparingly given, as it is apt to lead to the more extensive diffusion and absorption of the poison. But in some instances water has been found to be productive of great benefit, and has aided the efforts of the stomach to expel the poison by vomiting. (See the case by Dr. Brush, ante, p. 209.) The proper antidotes are chalk, compound chalk powder, magnesia or its carbonate, made into a cream with water, and freely exhibited; or the bicarbonate of magnesia may be at once employed. These remedies appear, from cases hitherto reported, to have been very efficacious when timely administered. A case in which this treatment was successful will be found reported by Mr. Tapson. (Med. Gaz. xxxi. 491.) The woman is said to have swallowed two ounces of the acid; but this is obviously a mistake. I agree with Dr. Christison in thinking that it is more likely to have been two drachms. A mixture of lime-water and oil might be advantageously employed. If much fluid has been swallowed, then the stomach-pump may be resorted to, and the stomach well washed out with lime-water. The poison in many instances acts with such rapidity, as to render the application of these remedies a hopeless measure. The exhibition of the alkalies,—potash, soda, or their carbonates, must in all cases be avoided: since the salts which they form with oxalic acid, are as poisonous as the acid itself. In the after-treatment (in the stage of collapse) warmth should be applied and stimulants exhibited.

[There is no doubt of the propriety of a sparing use of diluents in the primary treatment. Experience has shown that the same quantity of acid given in substance, or in solution, will produce the most rapid and poisonous effects in the latter condition.—G.]

CHEMICAL ANALYSIS.

In the simple state.—This acid may be met with, either as a solid, or in solution in water. *Solid oxalic acid:* It is seen more or less prefectly crystallized in four-sided prisms, in which respect it differs from all other acids, mineral and vegetable. The crystals are unchangeable in air. They are soluble in water, and even in anhydrous alcohol; readily in alcohol at 0·815. The solubility in water is variously stated. I have found some specimens much more soluble than others; and the conclusion from the experiments which I have made is, that the acid is soluble in from twelve to fourteen times its weight of water. If there be any adhering nitric acid about the crystals, they are much more soluble. It is worthy of remark that this solution, unlike that of some other vegetable acids, (tartaric and citric,) undergoes no change or decomposition by keeping.

The crystals of oxalic acid are liable to be mistaken for those of two other salts, namely, the sulphate of magnesia (Epsom salts,) and sulphate of zinc (white vitriol.) The chemical differences are, however, well marked. Oxalic acid, when heated on platina foil, is entirely volatilized, or only a very slight residue of impurity is left, while the sulphates of magnesia and zinc are fixed. If the three substances be dissolved in water, it will be found that, while the solution of oxalic acid is intensely acid,—that of the sulphate of magnesia is neutral,—and of the sulphate of zinc, very feebly acid. If a solution of caustic potash be added to the diluted solutions of the three bodies in water, those of magnesia and zinc yield white flocculent precipitates; that of oxalic acid remains unchanged. The most simple method of distinguishing them is by the taste. Oxalic acid is immediately indicated by the intense acidity of its solution.

For the further analysis of the acid, the crystals must be dissolved in distilled water: but should a suspected *solution* of the poison in water be presented for examination, it will be proper, after testing it with litmus paper, to evaporate a few drops on a slip of glass, in order to observe whether crystals be obtained. If there should be none, there can be no oxalic acid present. If *fine and distinct prisms* be procured, then it will be proper to proceed with the analysis of the solution.

Tests.—1. *Nitrate of silver.* When added to a solution of oxalic acid, it produces an abundant white precipitate of silver. A solution containing so small a quantity of oxalic acid as not to redden litmus paper, is affected by this test (see p. 216;) but when the quantity of poison is small, it would be always advisable to concentrate the liquid by evaporation before applying it. The oxalate of silver is identified by the following properties: 1. It is completely dissolved by cold nitric acid, by which it is known from the chloride, iodate and cyanide. If collected on a filter, thoroughly dried and heated on thin platina foil, it is entirely dissipated in a white vapour with a slight detonation. When the oxalate is in very small quantity, this detonation may be observed in detached particles on burning the filter previously well dried. 2. *Sulphate of lime.* A solution of oxalic acid is precipitated white by lime water and all the salts of lime. Lime water is itself objectionable as a test, because it is precipitated white by many other acids—as the carbonic, tartaric, phosphoric and arsenious. The salt of lime, which, as a test, is open to the least objection, is the sulphate. As this is not a very soluble salt, its solution must be added in rather large quantity to the suspected poisonous liquid. A fine white precipitate of oxalate of lime is slowly formed. This precipitate should possess the following properties:—1. It ought to be immediately soluble in nitric acid. 2. It ought not to be dissolved by the tartaric, acetic, or any vegetable acid. Unless these precipitates be obtained, and their properties, as above described, determined, it cannot be said that oxalic acid is present in the solution.

Objections to the tests.—A liquid may be highly acid, yield crystals of oxalic acid on evaporation, and yet neither of the above-mentioned tests will act. This may happen when nitric acid is present in rather large proportion. As a matter of precaution in a doubtful case, the whole of the solution should be evaporated to crystallization, and the crystals dried and re-dissolved in water, before performing the experiments. It may be urged that the nitrate of silver precipitates other acids. Thus it forms white precipitates with the muriatic, prussic, and iodic acids, but these precipitates are insoluble in cold nitric acid, and do not detonate when dried and heated. The tests gives only a slight turbidness with the tartaric and citric acids when highly concentrated, instead of the abundant milk-white precipitate which is produced in a solution of oxalic acid, even when considerably diluted. Besides, there is no common acid but the oxalic which is precipitated by sulphate of lime. The paratartaric or racemic acid is precipitated by the test; but this acid is so rarely met with as scarcely to form an exception. Paratartrate of silver does not detonate like the oxalate. One objection to *sulphate of lime* is, that it precipitates acid as well as neutral solutions of the salts of baryta and strontia; and an acid solution of muriate of strontia might somewhat resemble in this respect a solution of oxalic acid; but not to mention that there are numerous ways by which this kind of difficulty may be easily removed,—the sulphates of barytes and strontia are eminently distinguished from oxalate of lime by their rapid precipitation, and by their perfect insolubility in nitric acid.

It has been recommended to neutralize the oxalic acid by ammonia, or potash, before applying the tests. The tests then certainly precipitate the oxalic acid more rapidly and abundantly. The objections, however, to the results become more numerous; and although these are easily susceptible of being removed in the hands of a practised toxicologist, it must be confessed that just doubts might often be entertained of the accuracy of the inference drawn from them by those who are not experienced in analysis. If the poison be already neutralized, as under the form of oxalate of ammonia, we have no alternative but to apply them. But we must remember that, while the two tests above mentioned, nitrate of silver and sulphate of lime, are precipitated by very few *acids*, they are precipitated by numerous *salts*; and the precipitates possess properties, which it is sometimes difficult to distinguish from those of the oxalates of lime and silver. Thus the alkaline tartrates, citrates, carbonates, phosphates, pyrophosphates, borates when concentrated, and iodates, are precipitated by one or both of the tests, like the oxalates; and if chloride of calcium be used instead of the sulphate of lime, the alkaline sulphates would also be precipitated. It may be observed, however, that the recently precipitated tartrate and citrate of silver are distinguished from the oxalate, 1, by their being decomposed, when boiled, while the oxalate of silver retains its whiteness, and is not decomposed at a boiling heat, but is readily dissolved, forming a clear solution, if oxalic acid be in excess. 2. The dried tartrate, heated on platina, burns without detonation: it becomes incandescent, and leaves a solid residue of metallic silver in a white fungous mass. The dried citrate partially decrepitates, and leaves a grey fibrous residue of metallic silver. Thus, then, these salts differ from the oxalate, which is entirely dissipated by detonation. Again, sulphate of lime does not precipitate an alkaline tartrate or a citrate, unless the salts be highly concentrated, while an oxalate is precipitated by the test even in the most diluted state. If, therefore, any doubt exist respecting the nature of the salt, it should be diluted with water before adding the test. The dried precipitates also differ. The oxalate of lime is silently converted, when heated on platina, to carbonate of lime or caustic lime, according to the temperature. The tartrate and citrate undergo partial combustion, leaving a grey or carbonaceous residue.

Other tests might be used—as for instance, the Chloride of gold, the Sulphate of copper, and solution of Strontia; but they add no force to the evidence afforded by those above mentioned, and we may conclude that when we obtain from an acid solution, a solid acid substance crystallizing in well defined prisms,—these crystals remaining unchanged in air, being volatilizable without combustion; and giving, when dissolved in water, on the addition of the nitrate of silver and sulphate of lime, the results above described, there can be no doubt that the substance is oxalic acid. Additional tests may or may not be employed, but any evidence, short of this, should not, it appears to me, be admitted to show the presence of the poison.

In liquids containing organic matter. The process is the same, whether it apply to liquids in which the poison is administered, or to the *matters vomited*, or lastly, to the *contents of the stomach.* This poison readily combines with albumen and gelatin, and it is not liable to be decomposed or precipitated by these or any other organic principles; it is, therefore, commonly found in solution in the liquid portion, which will then have a greater or less acid reaction. Should the liquid be very acid, we must filter it to separate any insoluble matters; should it not be very acid, the whole may be boiled, if necessary, with distilled water, before filtration is performed. A small quantity of the liquid may now be tested by a solution of *Sulphate of copper.* This serves as a trial test: for oxalic acid is the only acid precipitated by it. If a greenish white precipitate is formed either immediately or in a few minutes, oxalic acid may be present; but if none be formed, then either there is no oxalic acid present, or it is in very small quantity. On no account are the tests for oxalic acid to be employed in liquids containing organic matter, since nitrate of silver is easily precipitated by such matters, although none of the poison be present. It must be remembered in using the sulphate of copper as a trial test, that it will not precipitate oxalic acid if this be mixed with a strong mineral acid. Should the liquid be highly coloured, it may be first boiled for some time with well-washed animal charcoal. After this it may be filtered and concentrated by evaporation. To the filtered liquid, acetate of lead should be added until there is no further precipitation; and the white precipitate formed,—collected and washed. If any oxalic acid were present in the liquid, it will exist in this precipitate under the form of oxalate of lead. There are two methods of separating oxalic acid from the oxalate of lead.

1. Diffuse the precipitate in water, and pass into the liquid for about two hours, a current of sulphuretted hydrogen gas, taking care that the gas comes in contact with every portion of the precipitate. Black sulphuret of lead will be precipitated; and with it commonly the greater part of the organic matter, which may have been mixed with the oxalate of lead. Filter, to separate the sulphuret of lead; the filtered liquid may be clear and highly acid. Concentrate by evaporation; the sulphuretted hydrogen dissolved in the liquid is thereby expelled, and oxalic acid may be ultimately obtained crystallized by evaporation on a piece of plate glass. If there were no oxalic acid present in the precipitate, no crystals will be procured on evaporation. If crystals be obtained, then they must be dissolved in water, and tested in the way above directed. As a modification of this test, and to shorten the process, I have adopted the plan of digesting the precipitate in hydrosulphuret of ammonia, taking care not to use too much of this compound. Oxalate of ammonia is obtained in the filtered liquid.

2. The second process consists in boiling the precipitated oxalate of lead in water, containing a small quantity of sulphuric acid (the proportion regulated by the quantity of precipitate) for half an hour. Sulphate of lead is formed, and oxalic acid is set free; this becomes mixed with any surplus sulphuric acid. Filter, and neutralize cautiously by ammonia; the liquid often becomes turbid from the precipitation of a small quantity of oxalate of lead suspended by sulphuric acid. Allow this to subside, and then test it with the nitrate of silver and sulphate of lime. The characters of oxalic acid, if it be present, are immediately brought out; the sulphate of ammonia, here formed simultaneously with the oxalate, does not in the least interfere with the application of the tests. I have used both of these processes in cases of poisoning, and have succeeded in detecting a small quantity of oxalic acid by them, in the contents of the stomach. In two cases there had been violent

vomiting,—one proved fatal in twenty minutes, the other in three quarters of an hour. Of the two processes, the first is the best adapted for obtaining crystallized oxalic acid; the second is the more expeditious for obtaining chemical evidence of the presence of the poison.

It may be observed, that oxalic acid may be separated from many kinds of organic matter by its ready solubility in alcohol at (0. 815.) In employing this agent it would be proper to evaporate the liquid, and digest the dry residue in hot alcohol.

Objections. If, in the course of this analysis, acetate of lead should give no precipitate with the liquid even when neutralized, then oxalic acid is not present in a quantity sufficient to be detected. If it should give a precipitate, still there may be no oxalic acid present. The medical jurist must remember, that the acetate of lead is precipitated by most kinds of organic matter, and by many mineral and vegetable acids and their salts. Thus, if he be operating on the contents of the stomach, the presence of Epsom salt, (sulphate of magnesia,) any alkaline sulphate, common salt (chloride of sodium,—any tartrate, citrate, phosphate, or carbonate, would occasion a white precipitate with the acetate of lead. The presence of the sulphuric, muriatic, tartaric, citric, acetic, gallic, or tannic acid, either free or mixed with any of the above-mentioned salts, would produce the same result. A mixture of vinegar and salt, or of lemon juice and Epsom salts, would give an acid reaction, and be precipitated by acetate of lead like the oxalic acid. Common London porter is acid, and is precipitated by all the salts of lead. The answer to any objection of this kind is, that the analyst does not decide on the presence of oxalic acid from the effect of acetate of lead on the suspected liquid; but from the action of the proper tests for the poison on the acid substance separated by sulphuretted hydrogen from the precipitate which is formed by the acetate. This latter is not a test, but merely a means of separation to enable us to apply the other tests with safety. The nitrate of lead may be substituted for the acetate. It has this advantage, that, unlike the acetate, it is not so readily precipitated by the tartaric, citric, or gallic acid. Orfila condemns the plan here recommended for the separation of oxalic acid, because the same results would be obtained if binoxalate of potash or salt of sorrel were present in the organic liquid. Sorrel is much used in soup in France, but not, so far as I know, in England: hence the objection is one of a national character. In order to remove the supposed difficulty, Orfila proposes that the evaporated organic liquid should be repeatedly digested with pure alcohol at common temperatures:—this, according to him, will dissolve the oxalic acid, but not the binoxalate, and thus the two may be separated. (i. 190.) It would be indeed unfortunate for medical jurists if they were obliged to rely upon this process. Alcohol, as pure as it can commonly be procured, will certainly acquire an acid reaction when digested on binoxalate of potash. Hence, by relying on Orfila's process, the analyst would be deceived, and would pronounce oxalic acid, as such, to be present, when the deceased might only have partaken of the supposed sorrel soup. In order to meet this objection, it appears to me that we should inquire whether it is likely that the deceased had eaten of sorrel?—what were his symptoms before death?—whether he died suddenly after partaking of some liquid or solid?—whether there were any peculiar post-mortem appearances in the fauces, œsophagus, or stomach?—whether the quantity of oxalic acid found in the stomach, would not be utterly incompatible with the hypothesis that it was due to the presence of salt of sorrel taken at a meal? A case of criminal poisoning by oxalic acid, is not likely to occur where an answer to one or more of these questions would not be obtained.

It is difficult to state the exact length of the period after death, at which we may expect to discover this poison in the contents of the stomach. Having on one occasion detected it in the contents of the stomach of a person who had been poisoned, I placed the liquid aside for about *five* weeks during the summer. On re-examining it at the end of this period, it had become highly putrefied, ammonia had been formed, and not a trace of the poison could be detected. Nevertheless, except when in very small quantity, and under exposure to extreme putrefaction, oxalic acid does not seem to be liable to disappear in contact with animal and vegetable substances. On the 19th of January, 1835, *forty grains* of oxalic acid were added to six ounces of a mixture composed of gruel, porter, and albumen. The liquid has been kept in a bottle loosely corked; and although upwards of *twelve years* have elapsed, the liquid is still acid, and oxalic acid may be readily detected in it. The stomach after death may contain no traces of the poison. This will happen when the case is protracted, vomiting has been urgent, or the stomach-pump employed. On the other hand, the poison may be present, but in an insoluble form, when lime or magnesia has been given as an antidote. White chalky masses may in this case be found adhering to the mucous surface of the stomach, or subsiding as a sediment in the liquid contents. The analysis required for oxalate of lime will answer for the oxalate of magnesia.

Delicacy of the tests for oxalic acid.—The following are the results of some experiments on the tests for oxalic acid. A solution containing 1-220th of a grain of the common crystallized acid had a moderately acid reaction; and in a minimum of water yielded readily a well-marked precipitate both with nitrate of silver and acetate of lead. When this proportion of crystallized acid was diffused through an ounce of water, *i. e.* 96,800 times its weight, the solution had no acid reaction, and the above-mentioned tests, when added, caused no perceptible change. The action of the tests is therefore, in this as in all other cases, materially affected by the proportion of water in which the poison is dissolved. When the quantity of crystallized acid was equal to the 176th part of a grain diffused through 9680 parts of water, there was no acid reaction, and the nitrate of silver produced a decided precipitate, while the acetate of lead caused only a slight milky appearance. The same quantity of crystallized acid was diffused through 1,000,000 parts of water. In this case there was no acid reaction. The nitrate of silver produced a slight opalescence, but the acetate of lead caused no perceptible change in the liquid.

In order that the acetate of lead should act effectually, therefore, the solution of the poison must be concentrated to the smallest possible bulk. With less than three grains of oxalate of lead, it would be difficult to obtain a solution of the poison fitted for testing. This quantity would correspond to only about *one grain* of crystallized oxalic acid.

Although the nitrate of silver is an exceedingly delicate test, yet a certain weight of oxalate of silver must be obtained, in order to demonstrate the true nature of the precipitate. Less than a quarter of a grain would hardly give satisfactory results; but this quantity would correspond to about one-eighth of a grain of crystallized oxalic acid.

Oxalate of Lime.—When the oxalic acid exists in an insoluble form, *i. e.* as white chalky masses of oxalate of lime, the following process may be adopted. The suspected oxalate, previously well washed, should be boiled for about twenty minutes, with an equal weight of carbonate of potash, in distilled water. A partial double decomposition takes place:—the undissolved residue containing some carbonate of lime, and the liquid some oxalate of potash. The liquid may be filtered, neutralized by nitric acid, and then tested with the tests already described for a soluble oxalate. If there be any desire to determine the nature of the alkali with which the oxalic acid is combined, some of the sediment obtained from the liquids, or scraped from the surface of the stomach, may be calcined on platina foil, when caustic lime or magnesia will be left, easily known from each other by their respective chemical characters.

Oxalic acid in organic substances.—I am not aware that oxalic acid has ever been distinctly found in a free state in the stomach and intestines, excepting in those cases in which it has been taken as a poison. Combined with lime, as oxalate of lime, it constitutes a well-known form of calculus found in the bladder. In certain diseased conditions of the system, or under the use of certain articles of diet, oxalate of lime is also freely passed in a microscopically crystalline condition with the urine. I once found traces of this salt in a calculus in the intestines, taken from the appendix vermiformis cæci, in a fatal case of perforation: hence in some instances it is produced in the human body, but it appears to become immediately neutralized and rendered harmless by the base lime with which it meets in the secretions. In two fatal cases of perforation of the stomach, reported by Mr. Crisp, it is stated that the fluid which had escaped from the stomach contained a small quantity of oxalic acid. There was no satisfactory evidence as to the mode in which this acid found its way into the organ, whether by morbid secretion or otherwise: hence it does not appear to me that the alleged discovery of the acid in these cases can offer any objection to the statement here made.

Oxalate of lime, it may be remarked, is a large constituent of many dry lichens; it also enters into the composition of certain substances used for food or medicine; *e. g.* the leaves and root of the rhubarb plant (*Rheum palmatum,*) sorrel, and turnips. The stalks of the leaves of the rhubarb-plant now constitute a very common article of food, and accidents are said to have arisen from its having been eaten in large quantity before the leaves were fully developed. The following case is quoted in the Medical Gazette from the American Journal of Medical Sciences. A family of four persons, after eating very freely of the leaves of the domestic rhubarb or pie-plant, boiled and served as "greens," were all of them shortly afterwards seized with severe vomiting. In one of these persons the attack was followed by gastritis; but the others recovered directly after the vomiting. (xxxviii. 40.) It is stated in the same journal, from an analysis by Dr. Long, that one pound of the plant yielded twenty-four grains of oxalic acid; but as this is combined with lime, it is not likely to exert a poisonous action. It is a question whether, in certain stages of its growth, the oxalic acid may not be united to potash as binoxalate: for it is not probable that the quantity contained as oxalate of lime, in a moderate portion of the leaves of the plant, would cause gastritis.

The exact proportion of oxalic acid present in the combined state in the leaves and stalks of edible rhubarb (*Rheum Palmatum*) has not been determined. The root has hitherto been chiefly examined. Mr. Quekett has obtained from the root of Russian rhubarb from 35 to 40 per cent. of its weight of oxalate of lime. (Pereira, Mat. Med. ii. 1179.) This is an enormous proportion. According to another analysis, the proportion was 11 per cent. Dr. Schossberger, who has more recently investigated the subject, states that with a large quantity of sulphate of lime, he found but slight traces of oxalate of lime. (Pharmaceutical Journal, January 1845.) Mr. Wilson has shown experimentally, that the oxalate of lime thus taken into the stomach is again rapidly excreted by the urine, and that it does not remain in the system. (Prov. Med. Journ. Sept. 2, 1846, p. 414; and Nov. 11, 537.) In an analysis of five ounces of the leaf-stalks of the edible rhubarb I was unable to detect, either in the decoction or in the undissolved residue, any distinct traces of oxalic acid or oxalate of lime. Admitting, however, that oxalic acid combined with lime is an occasional constituent of the plant in variable proportion, it is difficult to comprehend how this can form any objection to the inference from a medico-legal analysis of the contents of the stomach. Oxalate of lime can never be found in the stomach in a case of poisoning by oxalic acid, unless an antidote of lime has been given. But if an antidote has been used, there will be evidence from symptoms, and in this case the discovery of any portion of oxalic acid in the stomach after death, may perhaps not be at all material:—the fact of poisoning will be sufficiently apparent from other circumstances. To give this objection any sort of force, it is necessary to suppose that a person, after having swallowed an enormous dose of rhubarb, or eaten an enormous quantity of the leaves, is by a mere coincidence seized with symptoms resembling those of poisoning by oxalic acid, and dies—chalk mixture having been exhibited before death;—further, that on inspection, no appearances indicative of the action of oxalic *acid*, are found in the fauces, œsophagus, or stomach; and lastly, that the presence of an insoluble oxalate in the stomach, is of itself chemical evidence that the person has died from taking oxalic *acid!* Such a hypothetical case appears to me to carry with it its own refutation, in the facts which must necessarily accompany it.

Absorption.—Oxalic acid is supposed to enter the blood, and give to it a dark brown colour. In a case which proved rapidly fatal, where two ounces of the poison had been taken, I examined four ounces of blood taken from the vena cava: but not a trace of oxalic acid could be detected. Dr. Christison states that he did not succeed in detecting the poison in the blood when it had been purposely injected into the femoral vein of an animal which died in thirty seconds. These negative results may be explained by supposing that the poison is decomposed, or the means of separating it from organic compounds are not sufficiently delicate. In two cases, leeches have been killed by the blood drawn by them, from persons who were at the time labouring under the effects of this poison. This seems to render it probable that the blood is poisoned, and, indeed, Orfila states that he has succeeded in detecting it in the urine, although not in the solid organs. (i. 190.) According to Wöhler, it may be detected under the form of oxalate of lime in the urine of animals to which it has been administered. This fact should not be lost sight of by the medical jurist, as the oxalate of lime, although frequently found in certain states of disease, is not a normal constituent of urine. The microscope would here render great assistance. It is probable that, in acute cases, death is solely to be ascribed to the absorption of the poison, and its peculiar action on the blood.

On solid Substances.—When solid organic matters, such as cloth, paper, or linen, are impregnated with oxalic acid, proofs of this may be obtained by digesting the spots in water and applying the usual tests. Oxalic acid does not corrode these substances like a mineral acid, but it very slowly produces orange yellow spots with a red margin on black cloth, in which respect it differs from the other vegetable acids. This acid is sometimes used for erasing writing-ink in cases of forgery, but in general, traces of iron are left in the paper, and the erasure may be detected by wetting the paper with ferrocyanide of potassium.

QUANTITATIVE ANALYSIS.—The quantity of oxalic acid present in a measured portion of any mixture, may be best estimated by precipitating it entirely as oxalate of lead. For every hundred grains of the dried precipitate we may allow forty-two grains of crystallized oxalic acid. In some instances, it may be more convenient to precipitate it as oxalate of lime by means of chloride of calcium; the quantity of oxalic acid may then be estimated from the equivalent of this oxalate.

For an account of BINOXALATE of POTASH, see chapter on Alkaline salts; also Vegetable Irritants.—SORREL.

TARTARIC ACID.

Tartaric acid has been hitherto considered not to possess any poisonous properties; but a case has recently occurred, in which there was no doubt that it acted as an irritant, and destroyed life. The case referred to was the subject of a trial for manslaughter at the Central Criminal Court, in January, 1845. The accused gave the deceased, a man aged twenty-four, by mistake, *one ounce* of tartaric acid instead of aperient salts. The deceased swallowed the whole dissolved in half a pint of warm water at a dose; he immediately exclaimed that he was poisoned: he complained of having a burning sensation in his throat and stomach, as though he had drunk oil of vitriol, and that he could compare it to nothing but being all on fire. Soda and magnesia were administered with diluent drinks. Vomiting set in, and continued until death, which took place nine days afterwards. On inspection, nearly the whole of the alimentary canal was found highly inflamed. The accused admitted that he had made a mistake, and tartaric acid was found in the dregs of the cup. The jury acquitted the prisoner. (*Reg.* v. *Watkins.*) Dr. Mitscherlich has performed with this acid a series of experiments on animals, which tend to prove that it is not a very active poison. He found that while the animal was under the influence of the poison the respiration was accelerated, and it then became laborious and slow. Great debility was a very prominent symptom, and soon ended in paralysis, death being preceded by slight spasms. He considered this acid to be less poisonous than the citric. Half an ounce was administered to a small rabbit, and proved fatal in one hour; three drachms killed a similar animal in forty minutes; and two drachms, given to a middle-sized animal, produced no effects. In the fatal cases, it was not found to excite inflammation of the small intestines. Tartaric acid appears to enter into the blood, and to act by absorption, for Wöhler detected it as tartrate of lime in the urine of animals to which he had administered it. (Med. Times, Sept. 1845, 341.) Dr. Christison says that he has given to cats one drachm of this acid in solution, without apparently producing any inconvenience to the animal; and that a surgeon of his acquaintance had known six drachms of tartaric acid to have been taken by an adult, in mistake for carbonate of potash, without exciting unpleasant symptoms. (On Poisons, 227.)

TREATMENT.—The same as in poisoning by oxalic acid.

CHEMICAL ANALYSIS.—Tartaric acid in powder is known by the following characters. 1. When heated on platina foil it burns with a pale reddish coloured flame evolving a peculiar odour and leaving an abundant residue of carbon. 2. It forms an acid solution in water, which when moderately concentrated yields a granular precipitate with a few drops of caustic potash slowly added. (Bitartrate.) 3. When a few drops of the acid solution are evaporated on glass, it crystallizes in an irregular plumose form. 4. The solution is precipitated white by lime water, when the latter is added in large quantity;—the precipitate being immediately dissolved by an excess of the acid. 5. It gives no precipitate, or only a slight opacity with nitrate of silver, (thus known from oxalic acid.) 6. It is not precipitated by chloride of calcium. 7. When exactly neutralized by potash, and nitrate of silver is added, a white precipitate is formed, which is immediately blackened and reduced to the state of metallic silver on heating the liquid to 212°.

Organic mixtures.—If the acid be not discovered in the stomach in the state of powder or crystals, we may digest the contents in alcohol, in which the vegetable acid is quite soluble.

For an account of BITARTRATE of POTASH, see Chapter on Alkaline salts, (post.)

CITRIC ACID.

This acid may also act as an irritant poison in large doses, but in the common state of lemon-juice, which contains only from one to two per cent., it must be regarded as inert. I am not aware that there is any case on record in which citric acid has acted injuriously in the human subject.

The results of Dr. Mitscherlich's experiments with this acid tend to show that when administered to animals in large doses, it is a somewhat active irritant: he describes it as highly deleterious! Dr. Christison found that, as with tartaric acid, one drachm in solution might be given to a cat with perfect impunity; while Mitscherlich found that this quantity caused violent symptoms in a rabbit, but did not prove fatal: two drachms killed a large rabbit in an hour and a half, and a small one in half an hour: half an ounce proved fatal to a large rabbit in half an hour. The symptoms indicated a remote influence on the spinal marrow. Although there is no direct proof that the acid is absorbed, the blood undergoes a considerable change and becomes very fluid. In a quarter of an hour after injecting the poison into the stomach, there were spasms of the jaw, followed by opisthotonos, difficult respiration, and imperceptible pulse. The attacks of convulsions recur until the animal dies from pure exhaustion. (Med. Times, Loc. Cit.)

Chemical analysis.—It will be here necessary to state those characters by which the citric is known from other vegetable acids. 1. When the powdered acid is heated on platina foil it melts, burns with a yellow flame, and leaves scarcely any residue of carbon. 2. It forms an acid solution in water, which, when long kept, undergoes, like the solution of tartaric acid, a decomposition into a black flocculent mould. 3. However concentrated this solution may be, it yields no granular crystalline precipitate on adding to it a few drops of caustic potash. 4. It is only slowly precipitated by lime-water, and the precipitate is easily soluble in water and in an excess of the acid. 5. It is not precipitated by chloride of calcium, or nitrate of silver. 6. When exactly neutralized by potash, and nitrate of silver is added, a white precipitate is formed, which is not blackened (like the tartrate,) but only partially decomposed, and turned of a yellow colour on boiling. 7. On slowly evaporating a few drops of the solution of citric acid on a piece of glass, it crystallizes in a stellated form, the small prisms radiating from a centre.

Organic mixtures.—This acid may, like the tartaric, be separated from many organic compounds by its solubility in alcohol.

In the state of *powder*, citric may be distinguished from tartaric acid not merely by the action of heat, but by the difference in the results produced on boiling it with a small quantity of concentrated sulphuric acid. Citric acid is dissolved, and forms a light-brown or yellowish-coloured liquid; tartaric acid is immediately converted by carbonization to a black tarry liquid.

ACETIC ACID.

This acid has been generally excluded from the class of poisons. Common vinegar, which contains only five per cent. of acetic acid, has often been taken in large doses without injurious consequences. From the experiments performed by Orfila on dogs, and from one case which he reports as having occurred in the human subject, acetic acid, when concentrated, appears to exert an irritant action on the body. (Annales d'Hygiène, 1831, vi. 159; also Toxicologie, ii 198.) This is not more than we might have expected, seeing that the concentrated acid is highly corrosive. In the case referred to, the deceased, a young female aged nineteen, was found dying on the highway. She suffered from convulsions, and complained of pain in the stomach, and died in a very short time. On inspection, the stomach was found neither softened nor corroded, but its mucous membrane near the pylorus was almost black. The mucous glands were prominent, and the vessels were filled with black coagulated blood.

In the recent experiments of Dr. Mitscherlich, it was found that the animal (a rabbit) to which this acid had been administered suffered from feeble respiration, great debility and spasms, in which state it expired suddenly. One ounce of acetic acid (Pharm. Bor.) killed a large rabbit in seven minutes: half an ounce killed a small rabbit in eleven minutes: two drachms another in an hour and a half: and one drachm another in about four hours. The dose of half a drachm did not cause death. In his experiments with distilled vinegar, he found that six drachms killed a small rabbit in eight hours, and that half an ounce did not cause death. The tunica propria of the intestines was found softened and of a brownish-red colour. Strong acetic acid has a powerful local action, dissolving readily all the tissues, and causing a large effusion of blood in the stomach. When applied to the skin, it is well known to have a vesicating effect, and to soften and dissolve the albumen. When swallowed, it produces severe symptoms immediately, as in the following case, reported by Dr. Melion. A man swallowed by mistake a spoonful of strong acetic acid. He had no sooner swallowed it, than he threw himself on the ground and rolled about in agony. He took a large quantity of water. Dr. Melion found on his arrival that the mucous membrane of the mouth was quite white, and the patient complained of a severe burning pain in the chest, and abdomen, and a feeling of sickness. He could scarcely speak: the skin was covered with perspiration, and the pulse was very quick and small. Milk, carbonate of magnesia, and oleaginous drinks were given to him. There was free vomiting and purging, and the patient soon recovered. (Journal de Chimie Médicale, 1845, 654.)

TREATMENT.—Magnesia or its carbonates, mixed into a cream with water.

CHEMICAL ANALYSIS.—1. Acetic acid is known by its peculiar odour, which, if not perceptible in the cold, may be evolved on boiling the liquid. 2. It is entirely volatile, and leaves no solid residue on evaporation. 3. It is not precipitated by the acetate of lead, in which it differs from other vegetable and some mineral acids. 4. It is not precipitated by lime-water or any salt of lime, whereby it is known from the oxalic, tartaric, and other vegetable acids. 5. When diluted it is not precipitated by nitrate of silver or nitrate of barytes, and it is thus known from the muriatic and sulphuric acids. 6. When neutralized by potash it forms a salt highly soluble in alcohol and water, which yields acetic acid when boiled with diluted sulphuric acid.

Organic mixtures.—Acetic acid may be obtained by distillation, with or without the addition of sulphuric acid from these suspected liquids; but if operating on the contents of the stomach, it must be remembered that acetic acid is a natural constituent, although in small proportion, of the gastric juice,—that it may also be formed by acetous fermentation,—and that in the shape of vinegar it is a very common article of food.

VINEGAR, which may be regarded as an organic mixture containing but a very small proportion of acetic acid (five per cent.,) may be examined by distilling a portion, and testing the distilled liquid for the acid. Vinegar, as it exists in commerce, always contains traces of sulphuric acid. In general it is easily recognised by its odour. Pelletan observed, in the case of a young child, that the abuse of vinegar led to a thinning of the mucous membranes of the stomach; and Landerer remarked that the milk of a wet-nurse, who had been in the habit of taking large quantities of the Vinegar of Roses, became thin, very acid, and deficient in casein and butter. The child gradually wasted and died, and the woman herself suffered severely. (Heller's Archiv. 1847, 2 H. S. 185.)

FORMIC ACID.

The only experiments which have been made on the poisonous action of this acid are those of Mitscherlich. He found that half an ounce of the diluted artificial formic acid (containing seven per cent.) injected into the stomach caused considerable inflammation of this organ, as well as of the small intestines. One ounce of this diluted acid killed a small rabbit in two hours and a quarter: half an ounce killed a rabbit in nineteen hours: two drachms caused much uneasiness, but this dose did not prove fatal. The formic acid is most probably absorbed, but it was not detected in the urine. The blood in the large veins was found to have a brown colour. The acid appears to have a specific action on the kidneys, and to produce diuresis. (Med. Times, Sept. 1845, p. 342.)

Chemical analysis.—This is a liquid acid possessing a remarkable odour, sufficient to identify it. It exists in the red ant (*Formica rufa*,) and may be obtained from this insect by distillation. It is commonly seen as an artificial product, and is not likely to be used as a poison. It is highly volatile. It is known—1. By its peculiar odour. 2. By its acid reaction. 3. By its producing a white precipitate with solution of nitrate of silver, and by its reducing that salt to a metallic state on the application of a moderate heat. 4. It reduces at a boiling temperature the protosalts of mercury, the bichloride of mercury, and chloride of gold.

For the action of certain eliminating tests on the Vegetable Acids, see ante, page 125.

CHAPTER XX.

POISONING BY THE ALKALIES. POTASH, SODA, AND THEIR CARBONATES—SYMPTOMS—POST-MORTEM APPEARANCES—TREATMENT—AMMONIA AND SESQUICARBONATE OF AMMONIA (SAL VOLATILE)—CHEMICAL ANALYSIS OF AMMONIA—POTASH AND SODA—BARYTA—ANALYSIS—STRONTIA—ITS EFFECTS ON RABBITS—ANALYSIS—LIME A CAUSTIC POISON—ANALYSIS.

POTASH AND SODA.

The symptoms produced by potash and soda, when taken in a strong dose are so similar, that one description will serve for both. It must be observed, that cases of alkaline poisoning are extremely rare, and have been, I believe, hitherto the result of accident. The most common form in which these poisons are met with, is in the state of pearlash (carbonate of potash) and soaplees (carbonate of soda.) The patient experiences, during the act of swallowing, an acrid, caustic taste from the alkaline liquid, if sufficiently concentrated, excoriating the mucous membrane. There is a sensation of burning heat in the throat, extending down the œsophagus to the epigastrium. Vomiting is not always observed; but when it does occur, the vomited matters are sometimes mixed with blood of a dark brown colour, and detached portions of mucous membrane:—this effect depending on the degree of causticity in the liquid swallowed. The surface is cold and clammy:—there is diarrhœa, with severe pain in the abdomen, resembling colic. The pulse is quick and feeble. In the course of a short time, the lips, tongue and fauces, become swollen, soft and red.

POST-MORTEM APPEARANCES. There are strong marks of the local action of the poison on the mucous membrane of the mouth, fauces, and œsophagus. This membrane has been found softened, detached and inflamed in patches of a deep chocolate colour, sometimes almost black. The same appearance has been met with in the mucous membrane of the larynx and trachea. The stomach has had its mucous surface eroded in patches, and there has been partial inflammation.

PERIOD OF DEATH. The earliest fatal case which I have found reported, is that of a boy, who died in *three hours* after swallowing three ounces of a strong solution of carbonate of potash. In another case, which occurred at Yarmouth, in 1835, a child aged three years, took a small quantity of pearlash, which had deliquesced, and died in twenty-four hours. Death was caused in this instance by the inflammation induced in the larynx, causing an obstruction to the process of respiration. In this respect, the caustic alkalies may destroy life like the mineral acids. But death may be a slow result of these poisons. Thus, in an instance which was communicated to me, a lady swallowed by mistake, one ounce and a half of the common solution of potash of the shops, which contains about five per cent. of caustic alkali. She recovered from the first symptoms of irritation, but died seven weeks afterwards, from pure exhaustion, becoming greatly emaciated before her death. Orfila refers to two cases of poisoning by carbonate of potash, in each of which half an ounce of this substance was taken by mistake for aperient salts. The patients, two young men, recovered from the first effects; but ultimately died, the one three months, and the other four months, after the poison had been taken. The secondary fatal effects appear to be due to diarrhœa, great irritability of the stomach, loss of the functions of this organ from the destruction of the lining membrane,—and stricture of the œsophagus,—either of which causes may prove fatal at almost any period.

QUANTITY REQUIRED TO DESTROY LIFE.—The quantity of any of these alkaline poisons, required to destroy life, is unknown.

TREATMENT.—We may administer freely, water containing acetic or citric acid dissolved,—lemon-juice, or the juice of oranges. Demulcent drinks, as albumen, milk, gruel, or barley-water, will also be found serviceable. The free exhibition of oil has been found useful.

CHEMICAL ANALYSIS.—CAUSTIC POTASH and SODA are best known from their respective carbonates by giving a brown precipitate with a solution of nitrate of silver. The CARBONATES, on the other hand, yield a whitish yellow precipitate. Caustic *potash* is known from caustic *soda* by the following characters. 1. Its solution, when not too much diluted with water, is precipitated of a canary yellow colour, by bichloride of platina. 2. It is precipitated in granular white crystals, on the addition of an excess of a strong solution of tartaric acid, or by digesting in it a large crystal of tartaric acid. This test sometimes answers better by adding the alkali gradually to the acid, and occasionally agitating the mixture. Caustic soda is not precipitated by either of these tests, which will serve equally to distinguish the salts of potash from those of soda, if we except the binoxalate and bitartrate of potash, which, from being but little soluble in water are not precipitated. 3. If we neutralize the two alkalies by diluted nitric acid, and crystallize the liquid on a slip of glass, should the alkali be potash, the crystals will be in the form of long slender fluted prisms; if soda, of rhombic plates. 4. A fine platina wire may be dipped into the alkaline liquid, and then dried by holding it above the flame of a spirit-lamp. In this way a thin film of solid alkali is obtained on the wire. On introducing this into the colourless part of the flame, if it be potash, the flame will acquire a rose or lilac colour; if soda, a rich yellow colour. This test applies to the salts of the two alkalies. Care must be taken that the platina wire is perfectly clean. When the quantity of alkali or alkaline salt is large, the experiment may be performed in a platina capsule, alcohol being added to the salt and the mixture boiled.

The CARBONATES of potash are known from those of soda by the above tests. The CARBONATE is known from the BICARBONATE of either alkali, by the fact that the former yields immediately a white precipitate, with a solution of sulphate of magnesia, while the latter is unaffected by this test. It is important for the analyst to remember that caustic potash and soda, their respective carbonates, and the sesquicarbonate of ammonia, are often contaminated with oxide of lead, and give a black precipitate with sulphuretted hydrogen or hydrosulphuret of ammonia. This happens whenever the solutions of these salts have been kept in flint-glass bottles. (See ante, p. 118.)

In liquids containing organic matter.—Such liquids are frothy: they possess an alkaline reaction, a peculiar alkaline odour, and are unctuous to the feel. Potash and soda soften and dissolve most kinds of animal and vegetable matter. They also act upon woollen articles of clothing. If the alkali be *ammonia*, this will be announced by the odour, and it may then be obtained by distillation. If it be in small proportion, this can afford no evidence of poisoning; since many animal fluids contain ammonia, and in those which do not contain it, it is easily generated either by spontaneous decomposition, or sometimes even by the heat required for distillation. Should the alkali be in large quantity, this is no evidence of poisoning by it, unless we at the same time discover obvious marks of its local action on the mouth, fauces, œsophagus and stomach. If the organic liquid be highly alkaline, but give out no odour of ammonia, either by itself or on distilling a portion of it with caustic potash, the alkali may be either potash or soda, or their carbonates. The latter would be known by the liquid effervescing on adding a portion to an acid. The organic liquid may be evaporated to dryness, then heated to char the animal and vegetable matter, and the alkali will be recovered from it in the state of carbonate by digesting the residuary ash in distilled water. Any caustic alkali may be effectually separated by digesting the incinerated residue in pure alcohol. It has been also recommended to neutralize by muriatic acid, to evaporate, incinerate, and procure the alkali for analysis in the state of chloride. Mere traces of these alkalies furnish no evidence of poisoning, since all the animal liquids and solids yield soda, and many of them potash. In no case will the discovery of the alkalies be any proof of poisoning, unless the alkali be in *large* quantity, and the marks of its action be apparent in the fauces and stomach. According to the experiments of Orfila, potash is *absorbed* and conveyed into the blood. The alkali is eliminated by the urine, which is thereby rendered alkaline. When he gave about one drachm of potash to dogs, the presence of this alkali was detected after the lapse of six hours in the liver, spleen, and kidneys. Owing to the solvent action of this poison on fibrin and albumen, the blood, although it may be darker in colour, is never found coagulated in the vessels after death.

AMMONIA. SESQUICARBONATE OF AMMONIA.

The *vapour* of strong ammonia is poisonous. It may destroy life by producing violent inflammation of the larynx and pneumonia. It is often most injudiciously employed to rouse persons from a fit. A case is on record, of an epileptic having died, under all the symptoms of croup, two days after the application of strong ammonia, in this way, to the nostrils. A very singular case of recovery from the poisonous effects of this vapour, by Dr. Sanchard, will be found reported in the Annales d'Hygiéne (Janvier 1841.) The *solutions* of ammonia and its sesquicarbonate produce symptoms similar to those described in speaking of potash. The only difference observed is, that the sense of heat and burning pain in the throat; fauces, and stomach, are much greater. Cases of this form of poisoning are rare. Dr. Sanchard relates an instance which occurred in France, where a boy, only six years old, poisoned his younger sister by pouring several teaspoonsful of strong solution of ammonia down her throat. A case is likewise reported where a strong dose of the solution killed a man in *four minutes*. (Christison, 167.) In the following instance of poisoning by ammonia, the exact quantity taken was unknown, but the solution was sufficiently strong to act chemically on the mouth. A woman, aged twenty-four, swallowed about half a wine-glassful of mixture containing a large quantity of strong solution of ammonia put into it by mistake. She immediately fell backwards in a state of insensibility, and appeared as if choaked. When seen about six hours after the accident, she complained of severe burning pain down her throat and in the epigastrium, which was tender on pressure. There was great debility, the voice was reduced to a whisper, and the countenance expressed anxiety. There was also great difficulty of deglutition, the pupils were widely dilated, breathing difficult, the tongue coated with white fur, painful and tender; two or three patches of its mucous membrane peeled off, and there were convulsive twitches of the right arm. Diluted vinegar and other remedies were employed, but the patient did not entirely recover from the effects until after the lapse of ten days. On the fifth day, there were still great pain and tenderness in the epigastrium and left hypochondrium. (Case by Mr. Wilkins, Lancet, April 4, 1846, p. 385.) A recent case is referred to in the Journal de Pharmacie (Oct. 1846, p. 285,) in which from one to two drachms of ammonia, unknowingly administered, caused death. There was violent vomiting, with bloody stools; and, on inspection, blood was found effused in the intestines. There was also a remarkably fluid state of the blood in the body. In another instance, a man walked into a druggist's shop, and asked for a small quantity of ammonia to take some spots out of his clothes. The druggist poured about a teaspoonful and a half into a glass. The man suddenly swallowed it, and fell instantly to the ground. He soon afterwards died, complaining of the most excruciating pain. (Journal de Chimie Médicale, 1845, 531.)

SESQUICARBONATE OF AMMONIA.—The solution of this salt (sal volatile) is probably more active as a poison than is commonly supposed. The following case occurred to my knowledge in 1832. A man, in a fit of passion, swallowed about five fluid-drachms of a solution of sal volatile. In ten minutes, he was seized with stupor and insensibility; but, upon the application of stimulant remedies, he recovered. He suffered for some time afterwards from severe irritation about the fauces and œsophagus. These poisons are not often used by those who are intent upon suicide or murder, but there is one instance on record in which a man was tried for the murder of a child by administering spirits of hartshorn. (*Reg.* v. *Haydon*, Somerset Spring Assizes, 1845.) Of the action of the other salts of ammonia on man, nothing is known. (See page 234, post.)

CHEMICAL ANALYSIS.—The three caustic alkalies—potash, soda, and ammonia, are known from the solutions of the *alkaline earths* by the fact, that they are not precipitated by a solution of carbonate of potash. They all three possess a powerful alkaline reaction on test paper, which, in the case of ammonia, is easily dissipated by heat. AMMONIA is immediately known from potash and soda by its odour and volatility. If the solution in water be very dilute, the odour may be scarcely perceptible. The alkali may then be discovered, provided we have first assured ourselves by evaporating to dryness a portion of the liquid, that other alkalies and alkaline salts are absent,—by adding to the solution a mixture of arsenious acid and nitrate of silver. The well-known yellow precipitate of arsenite of silver will be instantly produced. The same result takes place when a carbonate (even bicarbonate of lime) is present; but if any carbonate or other salt existed in the liquid, it would be left on evaporation. In addition to these characters, ammonia re-dissolves the brown oxide of silver, which it precipitates from the nitrate, while potash and soda do not. For the detection of ammonia in *organic mixtures*, see p. 224. The SESQUICARBONATE OF AMMONIA may be known from other salts by its alkaline reaction, its odour, and its entire volatility as a solid:—from pure ammonia—1, by its effervescing on being added to an acid; 2, by its yielding an abundant white precipitate with a solution of chloride of calcium;—from the carbonates of potash and soda, among other properties—1, by its giving no precipitate with a solution of the sulphate of magnesia; 2, from the rich violet-blue solution which it forms when added in excess to the sulphate of copper; 3, by its odour and volatility.

BARYTA.

This substance is undoubtedly a poison, but very little is known concerning its action on the human subject. Pure baryta itself is a caustic alkali, which is not likely to be taken as a poison, seeing that it is rarely met with out of a chemical laboratory. The symptoms produced by baryta on the human subject, would be probably analogous to those caused by its soluble salts. These will be described in the chapter on poisoning by ALKALINE SALTS. This alkali, unlike potash and soda, is liable to produce symptoms indicative of an affection of the nervous system—as vertigo, convulsions, and paralysis. It is not, therefore, a pure irritant.

CHEMICAL ANALYSIS.—Baryta is a greyish-white substance, soluble in water: its solution possesses an alkaline reaction like potash, soda, and ammonia, and like them, it gives a brown precipitate with nitrate of silver; but it is known from these three bodies, by its being precipitated white by a solution of carbonate of potash. This last mentioned reagent equally precipitates lime and strontia, but among numerous other characters, baryta is known from lime by sulphuric acid, which precipitates baryta, but not lime. Sulphuric acid also precipitates a solution of pure strontia, but baryta is known by the action of a solution of sulphate of lime. This precipitates baryta immediately, but not strontia.

STRONTIA.

STRONTIA and its salts are not commonly regarded as poisons. I am not aware that any experiments have been performed with the pure alkali: but it appears from the observations of Professor Gmelin, of Tubingen, that two drachms of the CHLORIDE OF STRONTIUM, dissolved in water, had no effect upon rabbits. When the dose was raised to half an ounce, there was, in about five hours, paralysis of the extremities, with involuntary motions of the head: the animal died on the next day. On inspection, there was considerable ecchymosis, but no apparent inflammation of the mucous membrane of the stomach. Two drachms of this salt given to a dog caused vomiting, but no other bad symptom. Two drachms of the CARBONATE OF STRONTIA had no effect upon a rabbit, while only a drachm of the NITRATE affected the heart's action, and occasional diarrhœa. (Orfila, Toxicologie, i. 256.)

CHEMICAL ANALYSIS.—The analysis of strontia and its salts is fully given in the tables of alkaline poisons. It may be here mentioned that *Strontia* is a grey powder, soluble in water, the solution having an alkaline reaction and giving a brown precipitate with nitrate of silver. It gives a white precipitate with carbonate of potash. It is precipitated (more slowly than baryta) by sulphuric acid, in which respect it is known from lime: and it is *not* precipitated by sulphate of lime, in which respect it is known from baryta. There are other differences which may be derived from the tables. (See p. 126, ante.)

The soluble salts are characterized by their being precipitated, even in a very *diluted* state by sulphuric acid, oxalic acid, and sulphate of lime. They tinge the flame of alcohol of a deep crimson-red colour.

LIME.

This alkali has but little interest for the medical jurist: it is said to have destroyed life in one instance; and there is no doubt that lime, as well as its salts, may have an irritant action, but they have not the directly poisonous effects of baryta and its compounds. The following case of poisoning by lime is reported in Caspar's Wochenschrift. A boy, aged three years, while playing, tasted some slacked lime, and ate a considerable quantity of it. An emetic was given, and he brought up a mortary looking substance. The child became restless and feverish, had thirst, refused food, his lips were white, the mouth blackish, the abdomen hot and painful on pressure, and there were bloody evacuations. A few leeches were applied, and he recovered. (Med. Gaz. xxxv. 64.)

Chemical analysis.—The appearance of lime is familiar to most persons. In the caustic state, it is seen in masses of a white, greyish white, or brownish white colour. When slaked, the lime falls to a powder, and becomes whiter. It dissolves in acids without effervescence, if free from carbonate. It is soluble in water. The solution has—1, an alkaline reaction, and is precipitated by carbonate of potash; 2, it gives a brown precipitate with nitrate of silver (indicative of causticity;) 3, it is precipitated by oxalic acid, and the precipitate is insoluble in water, as also in an excess of the acid; 4, it is not precipitated by diluted sulphuric acid, in which respect it differs from Baryta and Strontia; 5, it is not precipitated by sulphate of lime; 6, when dissolved in muriatic acid, and the evaporated residue is boiled with alcohol, the flame is tinged of a crimson-red colour.

It may be observed that when the sulphuric and oxalic acids are used as tests, a solution of baryta is precipitated by the sulphuric and not readily by the oxalic acid (the precipitate being re-dissolved by the slightest excess of acid,) a solution of lime is precipitated by the oxalic and not by sulphuric acid, while a solution of strontia is precipitated by both acids.

In medico-legal analyses, the salts of these alkalies may be easily mistaken for those of baryta, as they are in many respects similar. Great caution should therefore be used, when a mixture alleged to be poisoned with a salt of baryta, is presented for examination. For a summary of the most remarkable chemical differences in respect to the action of liquid tests, see table of Alkaline Poisons, p. 126, ante.

CHAPTER XXI.

POISONING BY THE ALKALINE SALTS. BINOXALATE OF POTASH (SALT OF SORREL)—BITARTRATE OF POTASH (CREAM OF TARTAR)—BROMIDE OF POTASSIUM—IODIDE OF POTASSIUM— SULPHURETS OF POTASSIUM AND SODIUM—ALKALINE HYPOCHLORITES—MURIATE OF AMMONIA (SAL AMMONIAC)—NITRATE OF POTASH—SULPHATE OF POTASH—SULPHATE OF ALUMINA AND POTASH (ALUM)—CHLORIDE OF SODIUM (COMMON SALT)—SALTS OF BARYTA.

Some of the alkaline salts have been found to exert an irritant action on the system. The pure alkalies and their carbonates have a corrosive (chemical) action when concentrated, but they operate as irritants when diluted. The salts about to be described are not very energetic poisons, and, with one or two exceptions, require to be given in large doses in order to produce noxious effects.

BINOXALATE OF POTASH (SALT OF SORREL.)

The poisonous effects of this salt entirely depend on the oxalic acid which it contains. It is said to be much used for the purpose of bleaching straw and removing ink-stains—being sold under the name of essential salt of lemons. The smallest quantity retailed to the public is a quarter of an ounce, and for this, three-half pence is charged. Its poisonous properties are not commonly known, or no doubt it would be frequently substituted for oxalic acid. Three cases of poisoning by this substance have occurred within the last two years, two of which proved fatal, while in the other the patient recovered.

In the case of recovery a young lady, aged twenty, swallowed an ounce of salt dissolved in warm water. She was not seen by any one for an hour and a half: she was then found on the floor, faint and exhausted, having previously vomited considerably. There was great depression, the skin cold and clammy, the pulse feeble, and there was a scalding sensation in the throat and stomach. There were also continued rigors. Proper medical treatment was adopted, and she recovered in two days,—still suffering from debility and great irritation of the stomach. During the state of depression, it was remarked that the conjunctivæ were much injected, and the pupils dilated. There was also great dimness of vision. (Med. Gaz. xxvii. 480.)

In another of the cases,—a lady recently confined took by mistake half an ounce of the binoxalate, instead of cream of tartar. She had scarcely swallowed the draught, when she was seized with violent pain in the abdomen and convulsions: she died in *eight minutes*. On inspection, the mucous membrane of the stomach and small intestines was found inflamed. (Ann. d'Hyg. Avril 1842.) In the third case, a teaspoonful of this salt was taken for three successive mornings:—it produced severe vomiting; and about an hour after the third dose, the patient expired. There was no post-mortem examination.

We learn from these cases, that this salt is poisonous, destroying life almost as readily as oxalic acid itself; and that in the symptoms which it produces, it closely resembles that poison. In the second case, it destroyed life in so short a time as *eight minutes;* but probably the fatal effects were in this instance accelerated by the debilitated state of the person who took it.

Chemical analysis.—The *solid* salt is commonly seen in the form of a crystalline powder, or loosely crystallized in masses. It is not very soluble in water, easily taken up on boiling, but re-precipitated in great part on cooling. Its solution might be readily mistaken for oxalic acid; for, 1st, it has an acid reaction; and 2nd, it is precipitated by nitrate of silver and sulphate of lime, like oxalic acid; but with respect to the latter test, the precipitation, although more slowly produced, is much more copious. It is best known from oxalic acid—1. By its crystalline form, which, as seen in a few drops evaporated on glass, is plumose; and 2. by heating a portion on platina foil. While oxalic acid is volatile, the binoxalate leaves an ash, which, when sufficiently calcined, is alkaline, and it may be proved to contain potash by its dissolving in dilute nitric acid, with effervescence, and forming nitrate of potash. There is one vegetable salt for which it has been fatally mistaken, namely, the bitartrate of potash, or the cream of tartar. This latter is known by its solution not being precipitated by the sulphate or any salt of lime; and by its being rendered only milky or turbid on the addition of nitrate of silver. Lime-water furnishes a ready means of distinguishing these two salts. It precipitates both of them white, but the precipitate from the bitartrate of potash is re-dissolved on adding to it a small quantity of a solution of tartaric acid, while that from the binoxalate is not re-dissolved. It may be as well to mention another simple means of distinction, the colour of ink is immediately discharged by warming it with a few grains of binoxalate, but is unaffected by the bitartrate of potash.

In organic mixtures the process is the same as for oxalic acid itself. (See p. 214.) Although this salt is a natural constituent of sorrel, this vegetable is rarely used as an article of food in England, and then only in a small quantity. According to Mitscherlich, the proportion of binoxalate is only 0.75 per cent. of the weight of the fresh plant, or 3.75 per cent. of the juice; hence one ounce of fresh sorrel will yield but little more than three grains of the salt. The objection suggested by Orfila, that the salt found in the stomach, might be due to the presence of sorrel taken in soup, is therefore inadmissible, except where the salt is found in traces, and no symptoms of poisoning have existed. (See Sorrel, post.)

BITARTRATE OF POTASH (CREAM OF TARTAR. ARGOL.)

This salt has proved fatal in at least one instance, although it is not commonly regarded as a poison. The case is reported by Mr. Tyson; it occurred in this metropolis in 1837. A man, aged thirty-seven, took four or five tablespoonfuls of cream of tartar. He was seized with violent vomiting and purging. There was pain in the abdomen, thirst, feeble pulse, and the thighs and legs appeared paralysed. The fluid vomited was of a dark-green colour, and the motions had the colour of coffee-grounds. Death took place in about forty-eight hours. On inspection, the mucous membrane of the stomach and duodenum was found highly inflamed, the cardiac portion of the former being of a deep red colour, with some spots of black extravasation. The stomach contained a thick brown fluid, coloured by bile. The whole of the intestinal canal was more or less inflamed. According to Wöhler, this salt passes off by the urine, under the form of carbonate of potash, the secretion being alkaline.

Chemical analysis.—Cream of tartar is commonly seen in the form of a white powder. It is sparingly soluble in cold water, producing a slightly acid solution. If the *powder* be heated on platina foil, it is converted to carbon and carbonate of potash; the latter dissolves with effervescence in acids, and the nature of the alkali is thereby determined. The decomposition of the powder by heat, indicates that it is a vegetable acid salt. On adding the aqueous solution to lime water, a white precipitate is formed, which disappears on adding a further quantity of the acid solution. This proves that the acid is the tartaric. It is known from the simple tartrate of potash by its acidity, and by the fact that it is not precipitated by a salt of lime, while the tartrate is precipitated as a tartrate of lime. Chloride of platina gives no precipitate in the cold saturated solution.

In *organic mixtures*.—The salt being comparatively insoluble, may be found as a sediment at the bottom of the liquid. If dissolved, the liquid may be concentrated and alcohol added; cream of tartar is very insoluble in alcohol, and by this means the organic matter may be separated from it. If the liquid be strongly coloured, purified animal charcoal should be used to decolorize it. In searching for this substance in the stomach, it is proper to state, that it is a natural constituent of the potato. Belloc relates a case of alleged poisoning by Rochelle salt, the compound tartrate of potash and soda. The circumstances however render this statement somewhat doubtful. (Cours de Méd. Lég. 139.)

BROMIDE OF POTASSIUM.

But very little is known concerning the action of this salt upon the body. It operates as a diuretic, and affects the secretory organs, but not in so powerful a degree as the iodide. Of its poisonous properties the best account yet given is that published by Dr. Glover. (Ed. Med. and Surg. Journ. lviii. 338.) Barthez had ascertained some years previously, that when this salt was introduced into the stomach of a dog, in a dose varying from sixty to one hundred grains, it occasioned death if there was no vomiting; and on inspection the mucous membrane of the stomach was found inflamed. When injected into the circulating system it causes death; but this part of the subject is of little importance in a medico-legal view, since chloride of potassium and other equally inert bodies, have a noxious effect when thrown into the circulation. Dr. Glover has indeed found, that fifty grains of chloride of potassium, dissolved in half an ounce of water, sufficed to kill a dog when injected into the jugular vein.

The facts of most interest to the medical jurist are those which relate to the effects of the bromide of potassium when introduced into the stomach. In one of Dr. Glover's experiments, one hundred grains of the bromide, dissolved in three drachms of water, were injected through a flexible tube into the stomach of a strong rabbit. In five minutes the animal fell, and made ineffectual attempts to crawl. The breathing became difficult, and the action of the heart irregular. In about half an hour it was agitated with convulsions, became insensible, and died. On inspection the lungs were found congested; the stomach contracted towards the pylorus; the mucous membrane softened, grey, and dotted with numerous purple ecchymoses; there were two large bloody patches. The upper part of the small intestines presented similar appearances. The membranes of the spinal cord were congested, and there was some effusion of serum into the ventricles of the brain. Bromine (bromide of potassium) existed in the blood, the liver, and brain: hence there can be no doubt that the salt is absorbed.

These results were obtained with the pure bromide; but much of what is sold is impure. A drachm and a half of the impure salt was introduced by Dr. Glover into the stomach of a dog at mid-day. During the night the animal was violently purged. The same dose was given every day for a week: the animal became thinner, lost power over his limbs to some extent, but retained his appetite. Three drachms of it killed a rabbit in less than an hour and a half, with symptoms and appearances resembling those above described.

These results show that the bromide of potassium is by no means an energetic substance, and that it is only likely to be injurious to the human subject in very large doses.

Treatment.—The promotion of vomiting, and the exhibition of albuminous and mucilaginous liquids.

Chemical analysis.—The salt crystallizes in cubes, is fixed when heated on platina, dissolves in water, and forms a neutral solution. 1. The solution gives a light yellowish precipitate with nitrate of silver, not very soluble in ammonia, although more soluble than the iodide. 2. When chlorine is added to it, it acquires a yellow or amber colour; and upon then adding about one-fourth of its volume of ether and shaking the mixture, the ether rises to the surface, carrying with it the bromine in solution. Chloride of potassium is obtained on evaporation of the liquid. 3. A small quantity of the powdered salt heated in a tube with its bulk of peroxide of manganese and diluted sulphuric acid, yields bromine, known by the ruddy colour, odour, and bleaching properties of its vapour.

From *organic mixtures* it may be obtained by incineration and digestion in water.

IODIDE OF POTASSIUM (HYDRIODATE OF POTASH.)

This salt is extensively employed as a medicinal preparation, but it appears to have given rise, in some instances, to alarming symptoms, even when exhibited in small doses; and death has been said to result from its use. The following cases will perhaps best serve to illustrate its effects. A gentleman was ordered by his physician to take three grains of the iodide in a draught of peppermint water three times a day. After the third dose he felt very poorly; and an hour after the fourth dose he was attacked with a violent shivering fit, followed by headach, hot skin, intense thirst, quick and full pulse, vomiting and purging. These symptoms were succeeded by great prostration of strength. In spite of treatment, the purging lasted several days. The effects of the medicine in this case were so violent, although only *twelve grains* had been taken, that there is little doubt, if the patient had taken another dose, he would have been killed. (Med. Gaz. Sept. 3, 1841.)

In October 1841, a case was reported by Mr. Erichsen to the University College Medical Society, in which very alarming symptoms resulted from the exhibition of only *five grains* of iodide of potassium. There was great difficulty of breathing, discharge from the eyes and nostrils, inflamed conjunctivæ, and most of the symptoms of violent catarrh. The iodide was discontinued, and the patient recovered. Dr. Lawrie found that seven grains and a half of the iodide, in three doses, produced in an adult, dryness and irritation of the fauces, great difficulty of breathing, and other serious symptoms. In another case, thirty grains, in divided doses, caused severe headach and secretion of tears. In two instances, wherein he had prescribed it medicinally in small doses, it was, in his opinion, the cause of death. (Med. Gaz. xxvi. 588.) These cases, at least, show the necessity of caution in the medicinal use of this substance. The effects from small doses may, perhaps, be attributed to idiosyncrasy; still there seems to be good ground, from the results of experiments on animals, for ranking iodide of potassium among irritant poisons. It has not, so far as I know, caused death, if we except the two cases recorded by Dr. Lawrie. One drachm and a half of the solution has been taken by a young female without destroying life, although it produced very serious symptoms of irritation. (Devergie, Méd. Lég. ii. 536.) It has been suggested that the occasional adulteration of the iodide with carbonate of potash may account for the discrepant statements, as to its poisonous properties. In one instance, in which the medicinal dose had been carried to several drachms, the iodide was found to contain seventy-five per cent. of carbonate of potash!

Treatment.—There is no antidote to this poison. It should be removed as speedily as possible by the free use of emetics; and by the stomach-pump, if there be no excoriation of the mouth and fauces.

Chemical analysis.—Iodide of potassium is a white solid salt, crystallizing in cubes, like common salt. It is very soluble in water. In the *solid* state: mix with it a small quantity of peroxide of manganese, and moisten the powder with equal parts of strong sulphuric acid and water,—then heat the mass in a glass tube. The purple vapour of iodine is immediately evolved. If the tube be of small diameter, the quantity which may be thus easily detected is exceedingly minute. If the iodide be in *solution*, add to the clear liquid an equal part of a solution of starch, and then a few drops of strong nitric acid. The blue colour of the iodide of farina produced, will show that the salt is an alkaline iodide. This test is extremely delicate, and it may be usefully applied to the vomited matters to determine in a case of poisoning whether all the poison has been expelled. The potash may be detected by decomposing the salt at a high temperature with strong sulphuric acid, when sulphate of potash will result. This may be dissolved and tested.

In organic liquids.—If much coloured, boil with purified animal charcoal until the colour is in great part or entirely removed; then add to the liquid a solution of starch, and afterwards nitric acid. As a trial test, we may employ a slip of filtering paper soaked in starch, then dipped into the suspected liquid, and exposed to the fumes of nitric acid. In this case, the colour of the organic liquid does not interfere with the results. By this process, the iodide may be detected in the urine or saliva, when the analyst may not succeed in finding it in the contents of the stomach. If present in organic *solids*, we must dry them, incinerate them at a low temperature, and lixiviate the incinerated residue, when traces of the iodide may be detected in the filtered liquid by the addition of starch and nitric acid. The following is the result of an experiment. Ten grains of iodide of potassium were dissolved in six ounces of porter, mixed with an ounce of thick starch. The mixture was evaporated to dryness, the residue incinerated and lixiviated with one ounce of water. The solution was neutral. One drop, containing one-fiftieth of a grain of the iodide, gave a deep pink-red colour with starch and nitric acid.

Absorption.—Iodide of potassium, it is well known, undergoes absorption, and is carried into the secretions and all the soft organs, in which it may be detected by the above process, if not found in the stomach. The diffusion of this salt by absorption is strikingly indicated by the fact, that iodine was thus discovered in the liquor amnii of a female during parturition, who, for four months previously, had taken the iodide medicinally. (Comptes Rendus, 1845, i. 878.) In another case, Landerer detected iodine in the testicle of a man who had suffered from sarcocele for several years, and for the cure of which he had been in the habit of taking preparations of iodine. (Heller's Archiv. 1847, H. ii. 185.) This poison is chiefly eliminated in the urinary and salivary secretions. According to M. Bonjean, absorption is more complete in proportion to the smallness of the dose. He took a quarter of a grain in a large quantity of water, in divided doses, for a day, and he was able to trace iodine in his urine for seven days, and in his saliva for six days. He swallowed five grains of the iodide, dissolved in two ounces of water, and traces of iodine were discovered in the urine during twenty-eight hours, and in the saliva during seventeen hours only. During the whole day he suffered much from abundant salivation. (See Monthly Journ. of Med. Science, December 1845, p. 935.) The urine will generally be found to yield evidence of the presence of this salt most readily, and the process above described may be adopted for its detection.

SULPHURETS OF POTASSIUM AND SODIUM.

These compounds known under the name of *Liver of Sulphur*, are uncrystalline solids, of a red or red-brown colour. They form yellow solutions in water, which have an alkaline reaction, and are highly poisonous. No case of poisoning by them has occurred in England; but several fatal cases are reported to have occurred in France. The chief symptoms produced are—burning pain in the stomach, vomiting, and convulsions; the breath is tainted with the odour of sulphuretted hydrogen gas.

Symptoms.—The following case reported by Dr. Chantourelle, will illustrate this form of poisoning. A woman, aged forty, swallowed one morning by mistake a solution containing from three to four drachms of the sulphuret of potassium. She immediately perceived an acrid taste; vomiting ensued, by which a portion of the poison was ejected, and she became insensible. Dr. Chantourelle arrived in about eight minutes after the accident, but the patient was dying.

The air of the room was quite offensive, from the presence of sulphuretted hydrogen. The skin was livid, the tongue protruded, and a brown viscid saliva drained from the mouth. The eyes were fixed; the muscles had lost their contractility; the motion of the heart was scarcely perceptible; and there was slight convulsive inspiration occasionally. Death took place within ten or *fifteen minutes* from the time of swallowing the draught. On inspection there was a general congestion of the venous capillary system. No particular appearances were met with in the stomach or other viscera. The mucous membrane was pale, and covered with a layer of sulphur. (Galtier, Toxicol. i. 277.) It is not improbable that this patient died from the sulphuretted hydrogen gas passing into the lungs. In another instance, in which about the same quantity of sulphuret was swallowed, the patient experienced a sense of burning heat extending downwards to the stomach; vomiting followed, and the matters ejected smelt strongly of sulphuretted hydrogen, and contained, besides dark coagula of blood, a white substance intermixed, apparently precipitated sulphur. The other symptoms were coldness of surface, irregular pulse and severe burning pain in the epigastrium. Some violent after-symptoms manifested themselves of an inflammatory kind. These were not finally subdued until after the lapse of a month.

Among the APPEARANCES in fatal cases are a deep red colour of the stomach and duodenum, with blackness and liquidity of the blood. The sulphurets do not appear to be very active as poisons. They require to be taken in moderately large doses in order to produce serious effects: but, at the same time, they act with very great rapidity. In a case which occurred in April 1847, a lady swallowed by mistake two-thirds of a glass of a concentrated solution of sulphuret of potassium. Her sufferings were severe, and she died in three hours. (Med. Gaz. xxxix. 835.)

TREATMENT.—This should consist in the promotion of vomiting, and the administration of mucilaginous drinks, mixed with a weak solution of chloride, (hypochlorite) of soda or lime at intervals, so long as the breath exhales the odour of sulphuretted hydrogen. The chlorides decompose the poison, and set free sulphur, which exerts no injurious effect. The administration of acids would be attended with the injurious effect of evolving sulphuretted hydrogen more copiously. When, from the seat of pain, it is probable that the poison has descended into the intestines, it has been recommended to employ the chlorides of the alkaline bases, in the form of emollient enemata.

[Vinegar was formerly recommended, but is not to be relied upon. The best antidote, after vomiting, is common salt as advised by Dr. Chantourelle. Small doses of a solution of chloride, also act favourably.—G.]

CHEMICAL ANALYSIS. SULPHURETS.—When *solid*, sulphuretted hydrogen is abundantly evolved on adding diluted muriatic acid to the powdered sulphuret; and the alkali remains as a chloride. When in *solution*, subacetate of lead gives an intense black precipitate, by which the sulphuret may be identified in the smallest proportion. The PERSULPHURETS form rich amber-coloured solutions. They differ from the sulphurets in giving a copious precipitate of sulphur on adding acid, and in forming a brick-red precipitate with a solution of acetate of lead. For a modification of this test, see p. 175, ante—Sulphate of Barytes.

ALKALINE HYPOCHLORITES.

There are certain bleaching compounds which have been long known as chlorides of potash, soda, and lime, but which are now called by many chemists hypochlorites of the alkaline bases. The HYPOCHLORITE OF POTASH is commonly known as the *Eau de Javelle;* that of SODA as *Labarraque's liquid;* and that of LIME as *Bleaching powder.*

They may be regarded as irritant substances, but possessing only a weak action. In the following case, serious effects were produced by *Hypochlorite of Potash.* A female, aged seventeen, swallowed, at nine o'clock in the morning, a large glassful of the *Eau de Javelle.* No particular symptoms manifested themselves for a quarter of an hour, when the patient, having placed herself on the bed, was seized with convulsions, and then became insensible, in which state she remained for five hours. When brought to the hospital she had a burning pain in the fauces and œsophagus, extending to the stomach; difficult deglutition, with pain in the region of the larynx, epigastrium, and umbilicus; headach, and heat of skin. The urine was passed easily, but there was no purging. It was observed that the membrane covering the lips, and that lining the mouth and fauces, was much whitened, undoubtedly by the chemical action of the hypochlorous acid. Albumen was freely given to her; and this was followed by vomiting of white coagulated matter. Leeches were applied to the abdomen, and in the course of three days her health was completely re-established. (Orfila, Toxicologie, i. 234; Galtier, Toxicologie, i. 254.) In one instance a solution of this salt caused death. An aged man drank a quantity of the solution of hypochlorite, and died in sixteen hours. The lips, gums, and lining membrane of the mouth, were perfectly whitened. The mucous membrane of the pharynx was of a brownish colour, and covered with a thick layer of tenacious mucus. The membrane of the œsophagus was completely destroyed. Towards the cardia there was a dark eschar. The stomach itself was so softened, that it readily gave way with the slightest effort in its lesser curvature; and nearly the whole of the mucous membrane was thickened and converted into a dark sloughly mass. (Briand, Méd. Lég. ed. 1846, p. 447.) The Hypochlorites of Soda and Lime would probably, if taken in a large dose, act in a similar way.

Treatment. In the early stage the best means of treatment will be the free exhibition of albuminous and mucilaginous liquids, followed if necessary by antiphlogistic measures.

Chemical analysis. The hypochlorites of potash and soda are perfectly soluble in water; the hypochlorite of lime is only partially soluble. 1. The solution is colourless, strongly alkaline, and has the odour of chlorine. 2. It immediately bleaches a solution of sulphate of indigo. 3. It destroys the colour of litmus when an acid is added, or by exposure to air, in which case carbonic acid sets free the chlorine. 4. On adding sulphuric acid chlorine is evolved, easily recognised by its colour and odour. 5. When calcined they leave chlorides of metals, by the examination of which, therefore, the base is easily discovered.

Organic liquids. These compounds undergo decomposition in contact with most kinds of organic matter, especially with albumen, even in the form of mucous membrane. Galtier found that when hypochlorite of potash was mixed in equal parts with milk or coffee, the smell of chlorine had completely disappeared in about half an hour, and the presence of this gas could not even be detected by the addition of an acid. (Op. cit. i. 252.)

MURIATE OF AMMONIA (SAL AMMONIAC.)

This salt, according to the experiments of Orfila, acts as a poison on dogs, whether introduced into the stomach or the cellular tissue. It is absorbed, carried into the circulation, and acts specially on the nervous system, as well as upon the stomach. The mucous membrane was found in one case slightly reddened. Its effects on man are unknown, and probably it is not more energetic as an irritant than the chloride of sodium or potassium. (Toxicologie, i. 265.)

Treatment. The promotion of vomiting. There is no known antidote.

Chemical analysis. As a solid this salt is known by its tough fibrous character. 1. It is entirely volatile. 2. It is soluble in water, forming a neutral solution; but on boiling this becomes acid. 3. If moderately concentrated, the solution gives a yellow precipitate with bichloride of platina. 4. Muriatic acid is detected in it by the addition of nitrate of silver and nitric acid. 5. The ammonia is detected by boiling the solution with caustic potash, or heating the dry powdered salt with lime.

From *organic liquids* it may be obtained by filtration and slow evaporation. The salt may, however, be present in small quantities, as a result of the process of putrefaction.

NITRATE OF POTASH. NITRE. SALTPETRE.

This well-known salt is largely employed in the arts. It is an irritant, but only acts as such when taken in a large dose. It has destroyed life on several occasions. Its effects are, however, somewhat uncertain. An ounce, and even two ounces have been taken without causing very alarming symptoms. (Ed. M. and S. J. xiv. 34.) Dr. Bennett states that M. Gendrin was in the habit of giving it in doses varying from six to twelve, or sixteen drachms in the twenty-four hours without any dangerous symptoms resulting. (Med.-Chir. Review, April 1844, 549.) M. Mozade has more recently given it with benefit in cases of dropsy in from three to five drachm doses. (L'Union Médicale, 3 Juin, 1847, 274.) According to Tourtelle, no injury has followed even in cases where it was given in doses of an ounce. (Galtier, Toxicologie, i. 268.) Tartra denied that it had poisonous properties even in a very large dose, (op. cit. 135,) but cases have occurred which now leave no doubt on the subject. In one instance, quoted by Orfila, an ounce of nitre was taken by a lady in mistake for other salts. In a quarter of an hour, she suffered from nausea, vomiting and purging, and the muscles of the face were convulsed. The pulse was weak, the respiration laborious, and the extremities cold; but there was a sense of burning heat and severe pain in the epigastrium. She died in *three hours* after taking the dose. On dissection, the stomach was found highly inflamed, and the membrane detached in various parts. Near the pylorus, the inflammation had a gangrenous character. A large quantity of liquid coloured by blood was found in the stomach. (i. 283.) In another case, which proved fatal in sixty hours, where an ounce and a half of nitre had been taken, a small perforation was found in the stomach. (ib.) My friend, Dr. Geoghegan, of Dublin, has communicated to me the following case:—A man took from an ounce to an ounce and a half of nitre by mistake for salts. Severe pain in the abdomen followed, with violent vomiting, but no purging so far as could be ascertained. He died in about *two hours* after taking the salt. On examination of the body, a bloody mucus was found in the stomach,—the lining membrane was of a brownish red colour, generally inflamed, and in parts detached from the coat beneath. None of the poison could be detected in the stomach; but its nature was clearly established from the analysis of a portion left in the vessel which had contained the draught.

Two men swallowed, each, one ounce of nitre by mistake for Glauber's salt. They almost immediately experienced a sense of coldness in the course of the spine, trembling in the limbs, with violent vomiting and purging. The stools were bloody. They recovered in the course of a few days. (Casper's Wochenschrift, xviii. 1841.) A case is reported in the same journal, where one ounce of nitre killed a man in thirty-six hours.

A case recently occurred at Manchester, in which an old man, æt. 60, lost his life from an overdose of nitre which he had taken as a medicine. The dose amounted to about ten drachms: it caused profuse diarrhœa and death in about five hours. Death was referred to inflammation of the mucous membrane of the stomach and bowels, owing to the irritant action of the nitre. In a case reported by Dr. Letheby, a female, æt. 28, swallowed in two doses taken on two days, about an ounce of nitrate of potash. After the second dose, she was attacked with severe burning pain in the stomach, and violent vomiting followed by collapse. There was no purging, and the secretion of urine was stopped. The girl recovered in a few days. (Pharm. Journal, Feb. 1846, 356.)

In another case reported by Mr. Gillard, a man recovered in four days after having swallowed two ounces of nitrate of potash by mistake for Epsom salts. In about five minutes after taking the nitre, he felt a burning pain in his stomach, and this was immediately followed by sickness. Free vomiting was excited by mustard; and this probably led to his recovery. (Prov. Med. Journ. Aug. 19, 1846, 382.) These facts show how very uncertain are the effects of this saline compound.

Poisoning by nitre has been hitherto the result of accident. It is never taken for the purpose of suicide, the popular opinion being, that it is not poisonous; although the above cases show that it destroys life with greater rapidity than is commonly observed in the action of arsenic and corrosive sublimate. It is never likely to be employed by a murderer, since a dose sufficient to kill, could not be secretly exhibited.

Treatment. There is no antidote known. Vomiting should be freely promoted.

Chemical analysis.—See Nitric Acid, (ante, p. 188.) From the researches of Wöhler, it would appear that this salt is *absorbed* and eliminated in the urine. He detected it in the urine of a horse four hours after he had given to the animal, five ounces of nitre. M. Reynard also detected it in the urine of persons to whom it had been given medicinally. His plan consisted in throwing down the sulphates and phosphates of the urine by a solution of baryta; filtering, evaporating to dryness, and treating the residue with pure alcohol in order to dissolve out urea and other substances soluble in this menstruum. The nitre was then procured by digestion in water, evaporation, and crystallization. (Galtier, Toxicologie, i. 262.) Orfila states that he has detected nitre in the liver, spleen, kidneys, and urine of animals poisoned by it. (Ann. d'Hyg. 1842, ii. 434.)

SULPHATE OF POTASH.

This salt was formerly called *Sal de Duobus*, or *Sal Polychrest*. It has been regarded as inert, but of late years it has given rise to some important medico-legal investigations. A lady about a week after her delivery, took, by the prescription of her medical attendant, about ten drachms of the sulphate of potash in divided doses, as a laxative. After the first dose, she was seized with severe pain in the stomach, nausea, vomiting, purging, and cramps in the extremities. These symptoms became aggravated after each dose, and she died in *two hours*. It was supposed that some poison had been given by mistake; but that was not the case, and the question was, whether her death was or was not caused by the sulphate of potash. On inspection of the body, the mucous membrane of the stomach and intestines was found pale, except the valvulæ conniventes, which were reddened. The stomach contained a large quantity of reddish coloured liquid, which on analysis, was found to contain only sulphate of potash, and no trace of any common irritant poison. The examiners referred death to the sulphate of potash given in an unusually large dose, whereby it had acted as an irritant poison in a person whose constitution was already much debilitated. (Ann. d'Hyg. Avril 1842.)

The question whether this is to be regarded as a poisonous salt, of an irritant nature, has been much debated among members of the profession, owing to a case which was tried at the Central Criminal Court in October 1843. (The

Queen v. *Haynes.*) The prisoner had given the deceased, the night before her death, two ounces of sulphate of potash, dissolved in water; and it was alleged that she had a fortnight previous to this, taken, in divided doses, as much as a quarter of a pound of the salt. The woman supposed herself to be pregnant, which was disproved by an examination of the body; and it was charged that the prisoner had given her the salt with the intention of causing a miscarriage. After the last dose, she was seized with sickness, and died within a very short time. The stomach was found empty, but highly inflamed, and there was blood effused on the brain. One medical witness, referred death to the action of this salt as an irritant poison; the other to apoplexy, as an indirect result of the violent vomiting caused by it. The prisoner was acquitted of the charge of murder, but subsequently found guilty of the administering the sulphate with intent to procure abortion. Both of the witnesses admitted that, in small doses, the salt was innocent; but that in the dose of two ounces it would produce dangerous effects. A portion of the sulphate in this case was examined by Mr. Brande, as it was suspected that some poisonous substance might have become accidentally mixed with it; but it was found to be pure.

It is not improbable, from the symptoms and the inflamed state of the stomach, that the salt acted here as an irritant poison; and the fact of its being an innocent medicine in small doses appears to be no sound objection to this view; for the same circumstance is observed with respect to many substances, the poisonous or noxious properties of which cannot admit of dispute. Some have ascribed the irritant effects of this and other saline medicines—such as cream of tartar,—in large doses, to their insolubility, and to the fine spicula of the powdered salt acting mechanically upon the mucous membrane of the stomach. This explanation does not appear admissible: 1st, because some of these saline medicines, when taken dissolved—such as alum and nitre—have had a similar action; and, 2nd, the effects are very different, and far more rapidly fatal than in those cases where mechanical irritants—such as fine sand or iron filings—have been taken. In short, there is no doubt that if a similar quantity of the salt were taken perfectly dissolved in water, it would have an equally irritant effect; and sulphate of potash has been known to act in this way, when taken in divided and therefore in very soluble doses. A case in which it thus proved fatal in *two hours*, has been already reported (supra.) According to Mr. Mowbray (Medical Gazette, xxxiii. p. 54,) sulphate of potash is a salt much employed in France as a popular abortive. He quotes several instances in which, in large doses it produced severe symptoms, resembling those of irritant poisoning, and even death. In one case two drachms acted powerfully; and in another, that fell under his own observation, four drachms of the salt, administered to a lady after her confinement, had all the effects of an irritant poison. The case of *Haynes* is the first instance in which, I believe, it is publicly known to have proved fatal in England; and it shows that substances, commonly regarded as innocent, may give rise to important questions in toxicology.

There is no doubt that the most simple purgative salts may, under certain circumstances and when given in large doses, destroy life. A case has been already related (ante, p. 15) in which sulphate of magnesia caused death, and gave rise to a criminal charge in this country. It is said that sulphate of potash has in some cases caused vomiting and other serious symptoms, from its containing as impurity sulphate of zinc. This would be easily discovered by adding the ferrocyanide of potassium, which gives a white precipitate with a salt of zinc.

CHEMICAL ANALYSIS.—Sulphate of potash is easily identified. It is a dry hard salt, soluble in water, forming a neutral solution. This solution, if sufficiently concentrated, is precipitated both by tartaric acid and bichloride of platina, whereby potash is indicated; and the presence of sulphuric acid is known by the action of a salt of barytes. *Organic liquids.* This salt being insoluble in alcohol, may have the organic matter removed from it by treating the liquid containing it (previously concentrated) with alcohol:—or the substance containing the salt may be evaporated to dryness and incinerated, when the sulphate may be obtained by lixiviating the calcined residue with distilled water. The sulphate of potash exists naturally in some animal fluids, but only in minute traces.

SULPHATE OF ALUMINA AND POTASH. ALUM.

This substance is very commonly known; but it does not appear to have given rise to many accidents, at least in this country. One case of death from alum, appears in the Registration Report for 1838-9. A singular case occurred in Paris, in 1828, in which the alleged noxious properties of alum were brought into question. A lady swallowed a quantity of calcined alum dissolved in warm water, which had been supplied to her by mistake for powdered gum. The quantity taken was less than half an ounce. She immediately complained of a burning pain in the mouth, throat and stomach. She afterwards suffered from thirst, violent vomiting, and general disturbance of the system, from which she recovered in the course of two or three days. These effects were referred to the alum, and the party who supplied it by mistake was condemned to a severe punishment. On the case being carried to an appeal, Orfila contended that alum was not a poison; although he admitted that in the calcined state it was a caustic, and in order to establish his opinion of its inertness, he offered to swallow half an ounce on the spot! He referred to the symptoms under which the party laboured to some other cause; but on being further questioned, he admitted that a solution of calcined alum was likely to produce more serious effects than common gum, which the party should have taken. The punishment was mitigated. (Ann. d'Hyg. 1829, i. 234.) Orfila has since found that alum in a large dose, operates fatally on animals, destroying life in the course of a few hours! He states that he detected the salt in these cases in the substance of the stomach, liver, spleen and in the urine. (Ann. d'Hyg. 1842, ii. 433.) The reader will find a singular case of supposed poisoning by alum in the Ann. d'Hyg. 1832, ii. 180.

The SYMPTOMS produced by alum in a large dose, are frothing at the mouth, vomiting (the vomited matters containing alum,) purging, depression, weakness of the limbs, and the principal APPEARANCE is a reddish-brown colour of the mucous membrane, which may be found softened or disorganized, either wholly or in patches.

We cannot therefore refuse to admit the possibility of this substance acting as an irritant, on the same principle on which we admit the irritant properties of salts of a far more innocent character. It is, however, proper to observe, that this salt, given in large doses to animals, does not appear to affect them seriously unless the œsophagus be tied: *three drachms* have been taken at a dose, by patients, dissolved in six ounces of liquid, without any inconvenience resulting.

TREATMENT.—The promotion of vomiting and the free administration of calcined magnesia.

CHEMICAL ANALYSIS.—Common alum possesses a peculiar and astringent taste. It easily dissolves in water, forming an acid solution, which crystallizes on evaporation in regular octohedra. Its solution is not affected by ferrocyanide of potassium or sulphuretted hydrogen gas, whereby it is known from the true metallic saline solutions. Its sulphuric acid may be detected by a salt of barytes. On adding potash, a white precipitate of alumina falls down, which is redissolved by the addition of a larger quantity of the alkali. By this last character, it is known from the alkaline earths which are precipitated from their solutions by potash, but the precipitates are not redissolved. On adding ammonia in excess, alumina falls down. This may be separated by filtration, and on evaporating the liquid portion, and incinerating the saline residue, there will be found sulphate of potash. Calcined alum is a white uncrystalline substance, only partially soluble in water. About one-sixth is left as a residuary white powder, easily soluble in a mineral acid, and yielding alum by crystallization. The quantity dissolved by boiling water is, however, sufficient to allow its nature to be determined. From *organic liquids* it may be obtained by evaporation and incineration.

CHLORIDE OF SODIUM. COMMON SALT.

For the alleged poisonous properties of this substance, see ante, p. 14. These effects have been ascribed to the presence of iodide of sodium or arsenic, but pure salt may undoubtedly act as an irritant. It is exceedingly important in a medico-legal view to know that arsenic has been found in salt as a fraudulent admixture. (See Ann. d'Hyg. 1830, ii. 482; also 1832, ii. 288.)

Chemical analysis.—This has been already referred to in the description of the processes for detecting muriatic acid. On this account, as well as from the fact that it has already caused death when administered in a large dose, it may be proper to mention more in detail the chemical characters by which it may be identified. 1. It is easily dissolved by water, and a portion of the solution slowly evaporated on a slip of glass, yields well-defined *cubic* crystals. 2. It is insoluble in pure alcohol. 3. It yields abundant acid vapours with a kind of effervescence, when strong sulphuric acid is poured on it. These vapours form a dense white solid cloud, when a rod dipped in strong ammonia is brought near to them. 4. It yields chlorine gas when heated with equal parts of sulphuric acid, water, and peroxide of manganese:—the chlorine being recognised by its usual characters, (ante, p. 195.) About one-twentieth of a grain of the chloride may be in this way analysed, if the experiment be performed in a proportionably small tube. 5. The solution of the salt gives an abundant white clotted precipitate with nitrate of silver—possessing all the chemical properties of chloride of silver. (See ante, p. 195.) These properties of the precipitate must be positively determined, since there are numerous other salts which are precipitated white by nitrate of silver. These experiments, it will be perceived, merely indicate the presence of chlorine of muriatic acid. The characters of soda have been already given, p. 223.

SALTS OF BARYTA.

The principal salts are the Chloride, Nitrate, Acetate, and Carbonate, the last of which is insoluble in water. The sulphate, from its great insolubility is said not to be poisonous; but it would be well that this should be established by experiment, since insolubility is no criterion whatever of a substance being inert; although it is often erroneously assumed to be so, and the doctrine of chemical antidotes is chiefly founded on this view. Calomel and arsenite of copper are as insoluble as the sulphate of barytes, and yet are known to have a very powerful action on the body. (See ante, chapter on Antidotes.)

The only two preparations of baryta that have yet caused death, are the Chloride and the Carbonate.

CHLORIDE OF BARIUM.

One case of poisoning by this salt is reported by Wildberg. The symptoms were those of irritation, combined with an affection of the brain and nervous system. Vertigo, convulsions, and paralysis, have been remarked among them. In the case referred to, half an ounce proved fatal in two hours:—in another instance, one ounce taken by mistake for Glauber's salt destroyed life in *an hour*. In small doses even, the chloride has been found to affect the system powerfully. Orfila has satisfied himself that the chloride of barium is absorbed. He states that he has detected it in the liver, spleen, and kidneys of animals poisoned by it. (Ann. d'Hyg. 1842, ii. 217.)

Some years ago, this salt was discovered by Tiedemann and Gmelin in the urine and the blood of mesenteric veins and vena portæ, four hours after it had been given to animals. Hence there can be no doubt that it is absorbed, since baryta is not a natural constituent of the body.

CARBONATE OF BARYTA.

The *carbonate* of baryta is said to have destroyed life in two cases, in each of which only one drachm was taken; but the following case, which occurred to Dr. Wilson, shows that this compound is not so poisonous as the chloride. A young woman swallowed half a tea-cupful of the powdered carbonate, mixed with water, at a time when she had been fasting for twenty-four hours. There was no particular taste. In two hours, she experienced dimness of sight, double vision, ringing in the ears, pain in the head, and throbbing in the temples, with a sensation of distension and weight at the epigastrium. There was also palpitation of the heart. After a time she complained of pain in the legs and knees, and cramps in the calves. She vomited twice, a fluid like chalk and water. The skin was hot and dry, the pulse frequent, full, and hard. These symptoms gradually abated, and she recovered, although the pain in the head and epigastrium continued for a long time. (Med. Gaz. xiv. 448.)

ACETATE OF BARYTA.

The *acetate* of baryta would, no doubt, prove an active poison; but it is not much known.

TREATMENT.

The alkaline sulphates, either of soda or magnesia, should be exhibited freely in water; but, unless the patient is seen early, no treatment is likely to avail. The sulphates render the baryta less soluble, and certainly diminish, if they do not altogether destroy its poisonous properties. They will be of little service when the carbonate has been taken. In this case emetics and the stomach-pump may be used. As a chemical antidote to the carbonate, a mixture of vinegar with an alkaline sulphate may be employed. It would of course be improper to administer diluted sulphuric acid; and any other acid would render the poison more soluble.

CHEMICAL ANALYSIS.

The *soluble* salts of baryta possess these characters in common:—1. They are precipitated white by sulphuric acid or an alkaline sulphate, even when considerably diluted; those of lime are not precipitated, and those of strontia are very slowly precipitated when the respective solutions are diluted. 2. Diluted solutions of the salts of baryta are not precipitated by oxalic acid; those of strontia and lime are precipitated, even when much diluted with water. Oxalate of ammonia will throw down the salts of the three bases, unless the solution of the barytic salt be exceedingly diluted, when no precipitate is formed in it, or if formed, it is easily redissolved by oxalic acid. Solutions of the salts of strontia and lime, when equally diluted, are, however, readily precipitated by oxalate of ammonia, and the precipitate is not soluble in oxalic acid. 3. The powdered salts of baryta, when burned on a platina wire in the flame of alcohol, give a greenish yellow colour; those of strontia and lime give a rich crimson red. This experiment applies only to the soluble salts. The acids of the salts are known by their respective tests. The Chlorine of the *Chloride* of barium is known by the action of nitrate of silver, (p. 195, ante:) —the *Nitrate* of barytes, by precipitating the solution with sulphate of potash and obtaining nitre from the filtered liquid,—by at once adding to a saturated solution of the salt, copper filings and an excess of strong sulphuric acid, or by adding it in powder to the sulphate of narcotine, (p. 188, ante.) The *Carbonate* of baryta, by its becoming dissolved with effervescence in diluted nitric acid, and the action of the proper tests on the resulting soluble nitrate. Lastly, the *Acetate*, by boiling the solution with the diluted sulphuric acid, when acetic acid escapes, easily known by its odour. The solution of this last-mentioned salt is known from the other soluble salts of baryta by its being precipitated, when concentrated, by tartaric acid, in an excess of which it is soluble, as well as in a large quantity of water.

In organic liquids.—If the salt of baryta be dissolved, a good trial test is diluted sulphuric acid. This gives an abundant white precipitate should the poison be present in any quantity. We may then throw down the whole of the poison by an alkaline sulphate,—separate this by filtration, dry it and incinerate it with the organic matter with the addition of a little charcoal. In this way, it will be converted to sulphuret of barium, which may be decomposed by washing it with diluted muriatic acid, when chloride of barium, in a state fit for testing, will be obtained. It is proper to observe, that pure baryta and its salts are very apt to be confounded with subacetate of lead and other compounds of this metal. A clear distinction consists in this: the salts of lead are blackened by hydro-sulphuret of ammonia—those of baryta are not. Besides, sulphate of lead recently precipitated is easily soluble in concentrated muriatic acid, while sulphate of baryta is quite insoluble in this acid.

METALLOIDAL IRRITANTS.

CHAPTER XXII.

PHOSPHORUS—SYMPTOMS, AND POST-MORTEM APPEARANCES—ITS ENERGY AS A POISON—CHRONIC POISONING—CHEMICAL ANALYSIS. CHLORINE—IRRITANT PROPERTIES OF A SOLUTION OF CHLORINE. BROMINE—ITS EFFECTS ON ANIMALS. IODINE—SYMPTOMS AND POST-MORTEM APPEARANCES—TREATMENT—ANALYSIS. SULPHUR.

The metalloidal substances which here require consideration, are Phosphorus, Chlorine, Bromine, Iodine, and Sulphur.

PHOSPHORUS.

It is not often that we hear of cases of poisoning by phosphorus or its compounds. The following instance has been reported by Mr. Shephard, of Stonehouse. (Lancet, Dec. 1843.) A child, between two and three years of age, had been caught in the act of sucking and swallowing the heads of lucifer matches. Two days afterwards she appeared unwell,—there was some feverish excitement, but no active symptoms. The bowels were open, but the child did not suffer from pain, vomiting, or diarrhœa. Five hours after she was first seen, she became violently convulsed, and she died three hours afterwards. On inspection, a quantity of mucus mixed with blood, of a coffee-ground colour, was found in the stomach. The mucous membrane of the organ was very vascular throughout, and for the space of about two inches it had a florid-red colour, and was covered with mucus. There were no less than ten invaginations in the small intestines, many of which included from two to three inches of intestine, which was inflamed at the invaginated parts. There was no appearance of strangulation, and the bowels were empty. The medical opinion given at the inquest was, that phosphorus, in a finely divided state, was the cause of death, and a verdict was returned accordingly. More recently a female committed suicide by dissolving in vinegar the phosphorus from the ends of matches. She swallowed this mixture, and after undergoing the most severe suffering for eight days, she died labouring under symptoms resembling those of hydrophobia. (Journ. de Chim. Méd. 1846, 668.)

A case is reported in the Lancet (September 14, 1844,) in which phosphorus given as a medicine proved fatal to a boy aged ten years. The deceased had taken phosphorus in pills and in an oleaginous mixture for nearly four weeks. These were prescribed by a medical practitioner. When seen he was lying in a state of stupor, quite insensible, labouring under strong convulsions, hurried breathing, and a small pulse. He died some hours afterwards. The principal appearances were congestion of the brain, a bright vermilion colour of the anterior surface of the stomach externally, with softening of the mucous membrane within,—and the marks of violent irritation and inflammation of the muscular coats of the large intestines. The quantity of phosphorus taken was not stated, as it was given in divided doses. None had been given for ten days previously to death; nevertheless this was probably due to the long-continued use of this substance. The stomach contained two ounces of a coffee-ground liquid and a large quantity of mucus. A singular case is reported by Dr. Graff, in which a young woman swallowed the phosphorus contained on about three hundred matches, equal to rather less than *five grains* of pure phosphorus, and recovered from the effects. The symptoms do not appear to have been very severe, a fact ascribed by the reporter to the phosphorus having been in an intimate and probably insoluble state of combination with other substances in the matches. Mr. Shephard's case above related, shows that this explanation is inadmissible, and we must look upon this, admitting the case to be correctly reported, as a very remarkable instance of recovery. (Henke, Zeitschrift, 1842, ii. 283.)

SYMPTOMS AND EFFECTS.—So few cases of poisoning by phosphorus have occurred, that we are scarcely in a position to generalize upon its effects. It appears to be a powerful irritant poison, operating with some uncertainty, and it has been generally after many days that it has destroyed life. The symptoms are also exceedingly protracted in their appearance. Thus it is only after many hours, and sometimes one or two days, that signs of irritation and other alarming symptoms appear, among which convulsions and spasms have been remarked; but when these once come on, the case proceeds rapidly to a fatal termination. In the first instance the patient experiences a disagreeable taste resembling garlick, which is peculiar to this poison. The alliaceous odour may be perceived in the breath. There is an acrid burning sensation in the throat, thirst, severe pain and heat in the epigastrium, with nausea and vomiting continuing until death. The vomited matters are of a dark green or black colour; they have the odour of garlick, white vapours may be seen to proceed from them, and in the dark they may even appear phosphorescent. Diarrhœa is among the symptoms, and the stools have been observed to be luminous. The pulse is small, frequent, and scarcely perceptible.

Chronic poisoning by this substance is accompanied by cardialgia, frequent vomiting, sense of heat in the stomach, diarrhœa, tenesmus, pains in the joints, marasmus, hectic fever, and disease of the stomach, under which the patient may slowly sink. Phosphorus in small doses is said to produce strong aphrodisiac effects. This view is borne out by the facts collected by Dr. Hartcop. (See Casper's Wochenscrift, 21. Feb. 1846, p. 115.) Some interest has been lately attached to the chronic form of poisoning by phosphorus from the researches of Dr. Strohl and others on the effects of the *vapour* upon individuals engaged in the manufacture of phosphorus matches. It has been remarked that persons thus engaged have suffered from necrosis of the jaw, carious teeth, and suppuration. There has been also marked irritation of the respiratory organs, and bronchitis has frequently shown itself among them. These effects have been attributed to the respiration of the vapours of phosphorus, which are supposed, by becoming acidified, to act chemically upon the bones. The subject still demands inquiry; but a very good summary of the facts, by Dr. Beck, will be found in the American Journal of Medical Sciences for Oct. 1846, 525. A case in which pneumonia was considered to have been induced by these vapours, is reported in the Medical Gazette (vol. xxxix. p. 210,) and another well-marked instance of the serious local and constitutional effects of these acid vapours has been published by Mr. Wright. (Med. Times, Dec. 19, 1846, 224.)

According to M. Dupasquier, phosphorus in vapour has no specific poisonous action:—it merely irritates the lining membrane of the bronchial tubes, and this effect is soon lost by habit. When other and more dangerous symptoms supervene, he thinks they must be ascribed to the accidental presence of arsenic in phosphorus. (Journal de Pharmacie, Oct. 1846, 284; also, Gaz. Méd. Dec. 5, 1846, 946.)

Post-mortem appearances.—From what has already been stated, we may be prepared to find marks of irritation and inflammation in the stomach and viscera. The stomach has been observed to be much contracted. The mucous membrane will be found inflamed, and probably softened. Gastro-enteritis proceeding to gangrene is the result of the action of this poison. M. Worbe found the stomach perforated in three places in a dog which had been poisoned by a solution of phosphorus in oil. The intestines and even the flesh of animals poisoned by phosphorus, have the odour of garlick, and appear luminous in the dark. (Galtier, Toxicologie, i. 184.) In a woman who died while taking phosphorus medicinally, it was remarked that the whole of the viscera of the body were luminous; thus indicating the extensive diffusion of the poison. (Casper's Wochenschrift, 21. Feb. 1846, 115.) There is no doubt that this poison is *absorbed*, although death may commonly be attributed to the local changes which it produces in the body. (For a further account of the appearances, see Casper's Wochenschrift, 28. Feb. 1846, 135.)

Quantity required to destroy life.—That phosphorus is a very active poison, is proved by two cases quoted by Dr. Christison (138.) In one, death was caused by a grain and a half in twelve days; in the other, by two grains in about eight days. It is supposed to operate as a poison only by becoming converted to phosphoric acid; but this is disproved by the fact, that phosphoric acid is far less poisonous than phosphorus. (See Mineral Acids, ante, p. 198.) A person has been killed by a quantity of phosphorus (case supra) equal to less than *three and a half* grains of phosphoric acid, while fifty grains of phosphoric acid have been given to a rabbit without effect!

The fatal dose is liable to vary according to many circumstances. Galtier states that it is comprised between three-quarters of a grain and two grains, and that even a third of a grain has destroyed life; while persons have recovered, as in one instance referred to (p. 243,) from a dose of *five* grains. In a case reported by M. Worbe, and quoted by Orfila, the ascertained fatal dose was less than *a grain and a half*. The subject was a man aged 27. The phosphorus was melted in hot water, and thus swallowed. Three days previously he had taken less than half a grain (three centigrammes) without ill effects. The patient suffered from all the effects of irritant poisoning, and died in twelve days. It is worthy of remark, however, that no active symptoms showed themselves for several hours. (Toxicologie, i. 55.)

Dr. Hartcop mentions that an apothecary took by way of experiment one grain, the next day two grains, and the third day three grains rubbed with sugar. He was then seized with gastro-enteritis, and died in spite of every attempt to save him.

The smallest fatal dose which I have met with is in a case quoted by Galtier. A woman, aged fifty-two, took in divided doses in four days about six centigrammes, or less than *one grain*, of phosphorus dissolved. The largest dose taken at once, *i. e.* on the fourth day, is stated to have been three centigrammes (0.462 grain,) or less than half a grain. Symptoms of pain and irritation appeared, and the patient died in three days. The œsophagus, stomach, and small intestines, were found much inflamed. (Toxicologie, i. 87.) One quarter of a grain dissolved in oil, has been known to produce burning heat in the abdomen, vomiting, and diarrhœa.

Period at which death takes place.—The earliest period is in a case related by Orfila, in which a young man in weak health took a dose of phosphorus (the quantity unknown,) and died in *four* hours. The same author mentions a case in which death did not take place until after the lapse of seventeen days. In general, several days elapse before a fatal result takes place, and during this time the patient undergoes much suffering. This was observed in a young female who swallowed a quantity of phosphorus-paste intended for poisoning rats. She did not die until the fifth day. (Journal de Chimie Méd. 1845, 580.)

Treatment.—This should consist in the administration of albuminous or mucilaginous drinks holding magnesia suspended, as well as in the free use of emetics and purgatives. When the symptoms have once manifested themselves, it is difficult to arrest their progress, and there is no known antidote to the poison. The exhibition of oil would be decidedly injurious, as this dissolves and tends to diffuse the poison. The presence of phospherus in the matters vomited or passed by stool, is known by their luminosity in the dark.

Chemical analysis.—Phosphorus is easily known by its waxy appearance and its remarkable chemical properties. It is insoluble in water, and, therefore, lumps of it, when it has been swallowed in substance and speedily ejected, may be separated from the vomited matters or contents of the stomach and intestines by simply washing them. It melts at about 110°, and takes fire a little above this temperature in air. It is soluble in ether, alcohol, and the oils. Ether is a very good solvent of phosphorus; and this liquid might, therefore, be in some instances advantageously employed for separating the poison when in a finely divided state from organic substances. It burns with a bright yellow flame and a thick white smoke—pyrophosphoric acid. The contents of the viscera of persons poisoned by phosphorus have been frequently observed, when dried, to be luminous in the dark; and when a portion has been warmed, a thick white acid smoke has risen from free portions of phosphorus in the contents. No better test of the presence of this poison could be desired; and, according to Orfila, it will detect phosphorus when it forms only one-thousandth part of the mixture.

When any doubt exists, the suspected portions of phosphorus picked out from the contents may be boiled in one part of nitric acid and three parts of water for an hour or longer. This will transform any phosphorus to phosphoric acid, and destroy organic matter. Pyrophosphoric acid, in a syrupy state, will be obtained on evaporating the liquid. This may be tested by the process elsewhere described. (See Phosphoric acid, ante, page 198.) Phosphorus may be detected some days after death, even, when the body is in a state of decomposition. Dr. Neumann, of Strasburg, has related a very interesting case in which the poison was easily discovered in the bodies of a shepherd and his dog *fourteen days* after interment. A shepherd, after having eaten some beet-root soup, vomited several times, complained of thirst, intense pain in the abdomen, and died after two days' continual suffering. His dog, which had eaten some of the food, became unwell, and died in two hours. The man lived unhappily with his wife, and, from some suspicion as to the cause of death, the body of the deceased, as well as that of the dog, was ordered to be disinterred and examined. As they had been buried fourteen days, and the weather was warm, the bodies were in an advanced state of decomposition. It was impossible to draw any inference from the condition of the viscera. A portion of the soup of which the deceased and his dog had eaten, was procured and submitted to examination. A small portion was spread on an iron plate, heated to a moderate temperature. Portions immediately burnt with a yellow light and a thick white smoke. In addition to this, the soup had the smell of phosphorus, and was luminous in the dark.

A very elaborate analysis of the contents of the stomachs of the deceased and his dog was then made. Muriatic acid and chlorate of potash were employed for oxidizing the phosphorus and converting it to phosphoric acid. In this way phosphorus was detected in both; and, from the viscera of the man alone, Dr. Neumann procured 15.48 grains of phosphoric acid, equal to 6.78

grains of phosphorus—quite enough to destroy life. A portion of alum was also found in the stomach.

It appeared that the deceased had complained of the taste of the soup, and had eaten but little. The dog, however, had eaten a large quantity; and this might account for his more rapid death. The woman was tried for the crime, but did not confess her guilt. (Casper's Wochenschrift, Mai 31, 1845.) The deceased, in this instance, was destroyed by phosphorus as it is prepared and sold in the form of a paste for poisoning vermin. The use of this substance for such a purpose has become common in some places. It is particularly destructive to mice, rats, and other animals; and when thus employed as a substitute for arsenic spread on bread, or mixed with fat, it may easily give rise to alarming accidents, even when taken in small doses. The subject, is therefore, deserving of the consideration of the medical jurist.

CHLORINE.

Of the gaseous form of this poison it is unnecessary to speak at the present time, as it will be more appropriately considered in the chapter on Poisonous Gases. The experiments of Orfila have shown that a saturated *solution* of chlorine in water acts like an irritant poison, producing effects resembling those caused by the mineral acids. Hitherto it has only proved fatal to dogs, in large doses, and where the œsophagus had been tied. The odour of the gas would be perceptible in the breath and vomited matters.

TREATMENT.—If a large dose of this substance has been swallowed, the best remedy to employ is albumen, either in the form of white or yelk of egg. Magnesia may be also freely exhibited with mucilaginous liquids.

CHEMICAL ANALYSIS.—A solution of chlorine possesses a yellowish colour and a strong smell of the gas, which is evolved from it on boiling a portion. The gas is then identified by colour, odour, and its bleaching properties. It gives a white precipitate with nitrate of silver insoluble in nitric acid. If long kept, the solution always becomes acid: hence, in this case, it first reddens, and afterwards bleaches, infusion of litmus.

BROMINE.

The principal facts which have been ascertained with respect to the poisonous properties of bromine are derived from the researches of Dr. Glover. (Ed. M. and S. J. lviii. 130.) It is not merely an irritant, but a strongly corrosive poison; its corrosive properties being destroyed only by excessive dilution. Dr. Glover has remarked, that two series of effects are produced by the introduction of bromine in large doses into the stomach:—1. The local action of the poison, arising from its volatility and the vapour penetrating into the fauces, air-passages, and lungs, as also from the corrosive and irritant action which it produces on the stomach and intestines. 2. Other symptoms proceeding from its absorption,—such as coryza, sneezing, salivation, and difficulty of breathing. After death, the fauces, œsophagus, and stomach, were found extensively inflamed and corroded; the mucous membrane of the stomach gelatinized; the duodenum of a yellow colour, and retaining a strong odour of the poison; the mucous membrane itself being thickened and brittle. The poison was here given in a concentrated state; and it was found that two fluid-drachms killed a dog in about five hours and a half. Dr. Glover ascertained that bromine was more irritant when diluted that when pure; but it is then far less corrosive. Like the mineral acids, the state of concentration in which it is swallowed must be regarded, more than the absolute quantity taken.

The only account we have of its effect on man is in two experiments performed by M. Bietske on himself. On one occasion he swallowed a drop and a half of bromine in about an ounce of water. This caused heat in the throat, œsophagus, and stomach, with colicky pains. Two drops and a half of bromine, taken in about two ounces of mucilage, gave rise to nausea, hiccough, and an abundant secretion of mucus. (Orfila, Toxicologie, i. 75.)

TREATMENT.—The free administration of albumen. Orfila recommends starch, which acts more by its viscidity than by any antidotal property. The colour of the starch ejected will indicate whether any of the poison is still contained in the stomach, as it acquires an orange-red or yellow colour by contact with bromine.

CHEMICAL ANALYSIS.—Bromine is a dark-red liquid, exceedingly volatile, and forming a ruddy vapour like that of nitrous acid. It is much heavier than water, and easily sinks in it; it has a very peculiar odour, which is quite sufficient to identify it. It bleaches vegetable colours, and gives a yellowish white precipitate with nitrate of silver not so soluble in ammonia as the chloride.

If free in *organic mixtures*, the colour and odour would at once identify it, and the bromine might be easily obtained by distillation at a low temperature. It is soluble in water, alcohol, and ether. A saturated aqueous solution contains one part of bromine to 41.23 parts of water (Glover.) Ether is its best solvent. It may be thus separated from liquids by agitating them with their bulk of ether. If it exist as hydrobromic acid, or as a bromide, chlorine should be first passed into the suspected liquid. This causes a reddish colour by setting the bromine free; and it may afterwards be separated by ether. All the solutions of bromine are immediately rendered colourless by the addition of an alkali (potash), colourless salts being thereby formed and dissolved. On this fact is based one process for separating bromine from organic mixtures. The liquid should be rendered just alkaline by potash, evaporated, and the residue incinerated at a low temperature; the ash dissolved in distilled water, filtered, and chlorine passed into it. The bromine may be then separated by ether.

For the BROMIDE OF POTASSIUM, see ante, page 229.

IODINE.

This well-known substance can be more easily procured and administered as a poison than chlorine or bromine. Iodine appears to be an active poison, although somewhat capricious in its effects. It is also an accumulative poison; for it may be taken for a long period in small doses without producing any serious results; but these may suddenly and unexpectedly follow and endanger life. In no instance, so far as I can ascertain, has this poison been employed for the purpose of murder; but in two instances of suicide it has been the cause of death.

Iodine has a strong irritant action in a state of vapour. M. Chevallier suffered from violent colic in consequence of his having respired it; and M. Lugol has observed that the vapour from iodized baths has produced symptoms of poisoning. I have myself experienced from the evaporation of and exposure to this vapour, heat and dryness in the fauces, with irritation of the conjunctivæ, lasting several days. Some of its effects are due to local irritation; others to absorption.

SYMPTOMS.—From experiments on animals, as well as from observation of its effects on man, iodine appears to have a strong local action as an irritant on the alimentary canal. In large doses, it occasions severe pain in the abdomen, with vomiting and purging; the vomited matters having the peculiar odour of iodine, and being of a yellow colour except when any farinaceous food has been taken, in which case they are blue, or even black. The fæcal matters may also contain iodine if the poison has been taken in the solid state. Besides these symptoms, there is great thirst, with anxiety, tremors, convulsions, cephalalgia, vertigo, motions of the extremities, and syncope; these last symptoms indicating that the poison has become absorbed. When taken for some time in small doses, it gives rise to salivation, vomiting and purging, pain in the epigastrium and cramps; the pulse becomes small and frequent; there is general wasting of the body; and it has been observed that, in this form of chronic poisoning, certain glands are liable to become affected and diminished by absorption,—as the breasts in the female, and the testicles in the male. Iodine produces these secondary effects (iodism,) whether it be taken internally or applied externally.

Post-mortem appearances.—As most of the individuals who have taken over-doses of iodine have recovered, it is possible to give only a summary of the appearances which may be probably met with. The stomach was found, in one fatal case, inflamed and excoriated: near the pylorus it was corroded, and the mucous membrane was detached for two or three inches. The lining membrane of the œsophagus and intestines was also inflamed. In Orfila's experiments on dogs, it was observed that the mucous membrane of the stomach and upper part of the small intestines was covered with a layer of yellow-coloured mucus: near the cardia there were small circular ulcers, surrounded with yellow areolæ, with yellow and brown patches scattered over the membrane, especially near the pylorus. When the layer of mucus was removed, the membrane beneath was found in an inflamed state, and the coats were softened.

Quantity required to destroy life.—Experience does not allow us to fix this with any precision. Orfila found that about one drachm of solid iodine sufficed to kill in a few days dogs, of which the œsophagus had been tied; but when this operation had not been performed, it required from two to three drachms to destroy life.

When exhibited medicinally for several days, in doses varying from three-quarters of a grain to two grains and a half, it produced emaciation, depression, great appetite, thirst, restlessness, frequent pulse, dry cough, and aphrodisiac effects. Orfila himself swallowed about a grain and a half of solid iodine while fasting, and he only experienced from this dose an unpleasant taste and nausea. The following day he took *three grains*, and he *immediately* perceived a sense of constriction and heat in the throat, which lasted for a quarter of an hour. This was followed by vomiting of a yellow-coloured liquid, in which iodine was easily recognizable. There was no constitutional disturbance, with the exception of a feeling of oppression, which lasted throughout the day. The next morning he swallowed, while fasting, *four grains and a half* of solid iodine; he experienced immediately the same heat and constriction in the throat, with nausea, eructations, salivation, and pain in the epigastrium. In ten minutes there was severe bilious vomiting with slight colicky pains, which continued for an hour. These were subdued by emollient amylaceous injections. The pulse became more frequent, the skin hot, and the urine high coloured; respiration scarcely affected. These symptoms yielded to the use of diluents and emollient injections, and the day following there was merely a sense of weariness. (Toxicologie, i. 67.) Orfila did not carry his experiments beyond this dose.

Nevertheless, there are some anomalies with respect to the action of this substance which appear inexplicable, unless we refer them to idiosyncrasy, or to a neutralizing influence exerted by the presence of food in the stomach. If iodine be taken on a full stomach, it is far less likely to act injuriously; and according to some, if farinaceous matters be present, its activity as a poison becomes lessened, although not effectually destroyed, by combination. It is also possible that a larger dose of iodine may be borne in a state of saline combination than when it is free; its local irritant action appears to be lessened under these circumstances. M. Sandras gave to a female a dose of eight grammes of tincture of iodine, containing *nine grains and a quarter* of this substance, without producing any symptoms of poisoning, and in this case the urine and fæces were coloured yellow by iodine. (Galtier, Toxicologie, i. 100.) In another instance, a still larger dose (ten grammes) of the tincture was given, and it caused dryness of the fauces extending to the epigastrium, severe pain in the stomach, and violent attempts at vomiting. In about an hour, the face was flushed, the pulse small and contracted, and there were slight convulsions. In about nine hours, these symptoms yielded to the use of warm water, given to promote vomiting, and opiate remedies. (Orfila, Toxicologie, i. 67.) Dr. Christison quotes the case of an infant, only three years old, who took three drachms of the tincture at once, and suffered only from attempts to cough, some retching, and much thirst. (On Poisons, 195.) The quantity of iodine here taken is not stated. Magendie states that he took ten grains of iodine in the form of tincture without injury.

These facts lead to the inference, that the poisonous properties of iodine are greater when the substance is administered in a free state than when it is taken dissolved in alcohol—contrary to what might be supposed. There is, however, a deficiency of cases with regard to the effects of iodine in the solid state. Judging from the effects of the largest dose taken by Orfila, from *fifteen to twenty* grains of solid iodine might, under circumstances favourable to its operation, endanger life.

The following fatal case of poisoning by the *tincture of iodine*, or a solution of iodine in alcohol, has been lately communicated to the Sheffield Medical Society by Mr. J. H. Smith. The patient, a woman, æt. 31, was suffering from erysipelas of the face: compound decoction of aloes was given, and the tincture of iodine ordered to be applied to the face with a brush. The quantity of the tincture sent, was one ounce, containing rather less than *one drachm* of iodine in one ounce of spirit. This tincture the patient drank instead of the mixture, and immediately exclaimed that she had swallowed poison. The tincture was given by a half-intoxicated man, for the mixture, although both bottles were marked with printed labels. Mr. Smith visited her very shortly afterwards, and found her complaining of a violent burning pain in the throat and stomach, followed by retching and slight vomiting; pulse rapid and full; eyes prominent and suffused. Mr. Smith immediately made her drink freely of a solution of bicarbonate of potash in warm water: he thought this might be beneficial by converting the iodine into iodide of potassium, which would be less irritating, and at the same time act as an emetic. Copious vomiting quickly followed, but without any relief to the symptoms. Some linseed tea was then administered, and ordered to be taken freely. When visited in a few hours, the symptoms continued unabated. Next morning, the pain was considerably relieved, but symptoms of depression succeeded, and she died the day following, about sixty hours after taking the poison. There was no post-mortem examination, as the coroner did not require it, and the friends would not allow it. (Prov. Jour. June 30, 1847, p. 356.)

Treatment.—This should consist in the promotion of vomiting by the free exhibition of warm water, albuminous, mucilaginous, or amylaceous liquids. Although farina or starch cannot be regarded as a chemical antidote, it is highly probable that it may act by diminishing the local irritant effects in suspending the particles of poison. It will also serve as an admirable test for determining whether the poison has or has not been entirely expelled from the stomach; since, when the liquid ceases to acquire a blue colour, we may infer that there is no more poison in the organ. To lessen irritation, opiates may be given, and, if necessary, antiphlogistic measures adopted. If the stomach-pump be used, a weak solution of carbonate of soda or potash may be employed. This will convert the iodine to a salt, and aid in dislodging any particles of the substance which may be adherent to the mucous membrane. When there is much local irritation, as in poisoning by tincture of iodine, the use of this instrument is inadmissible. In this case the free use of carbonate of soda should be resorted to, followed by emetics.

CHEMICAL ANALYSIS.—Iodine is a crystalline solid, of an iron-grey colour. When gently heated, it is entirely converted to a rich violet-coloured vapour, having a peculiar odour, by which it may be at once identified. It is soluble in water, alcohol, ether, and the bisulphuret of carbon. I have found that 7040 parts of water slowly dissolve one part of iodine—this is in the proportion of about one grain to sixteen ounces of water. If iodide of potassium, or any alkaline chloride, be dissolved in the water, iodine is rendered much more soluble. The aqueous solution is of a deep yellow-brown colour: it has the odour of iodine, and gives a rich blue colour (iodide of farina) with a solution of starch.

The aqueous solution is a useful test in post-mortem examinations, in order to determine whether any farinaceous articles of food are present in the stomach, or have entered into the last meal taken by the deceased. I have found the delicacy of this reaction to be such, that 1-1400th part of a grain diffused through 77,000 parts of water gave with starch a light *pink* colour; and the colour became *blue* when the proportion amounted to 1-470th of a grain in 28,000 parts of distilled water. The colour of the aqueous solution of iodine is immediately destroyed by one or two drops of hydrocyanic acid, or by the addition of any alkali or alkaline carbonate, in which case the iodine becomes acidified. Solid iodine stains all kinds of organic matter, whether skin, mucous membrane, linen, or paper, of a yellow-brown colour. These stains are immediately removed by potash, or any alkali.

Organic mixtures.—Iodine will give a blue, green, or dark colour to most organic liquids; it will also impart to them its peculiar odour. If the poison be in the solid state, owing to its great specific gravity (between three and four times the weight of water,) crystalline masses may be obtained by dilution with a large quantity of water. If the vessel be agitated, and the lighter and more easily suspended substances be suddenly poured off, the iodine will be found at the bottom. It is only when the fluid portion of the contents are of a yellowish colour that we are likely to detect it in a free state. Ether has been recommended as a means of separation by its superior affinity for iodine, and its sparing solubility in water. It combines with the iodine, dissolves it, and floats with it on the surface of the water. Orfila advises the use of bisulphuret of carbon, in which case the iodine is dissolved by this liquid, and carried down to the bottom of the vessel, forming a rich crimson-coloured solution. I have found this to be an excellent test for the presence of iodine, and an easy method of separating it from some kinds of organic liquids. The solution of iodine in the bisulphuret may be obtained by filtration through a wet filter, and the iodine subsequently procured from the bisulphuret by allowing the latter to evaporate spontaneously. (Toxicologie, i. 69.)

If iodine cannot be procured by these processes, owing to its intermixture with organic matter, two plans for its separation are still open to the analyst. The one commonly recommended consists in making the liquid slightly alkaline by potash, evaporating and incinerating the residue at the lowest possible temperature, in order to obtain the iodine as iodide of potassium. The saline residue should be dissolved in water, filtered, concentrated by evaporation, and mixed with a solution of starch. Nitric acid is then added, and the production of a blue colour indicates the presence of iodine. If the organic liquid be free from colour, then it may be at once evaporated, any acidity having been previously removed by potash.

In applying this mode of testing, it is necessary to remember that small quantities of iodide of farina are soluble in a strong solution of starch, and the pink or blue colour produced by a small portion of iodine may be thus concealed; hence, too much starch must not be added before the employment of nitric acid. Chlorine is sometimes recommended; but it is objectionable because, in a slight excess, it destroys the blue colour; and when the quantity of iodide obtained is very small, it may cause a failure of the experiment. This plan may be successfully employed for the detection of iodine in the blood and in all the secretions; for this substance is speedily absorbed, and carried to every part of the body.

A second method of acting on organic mixtures is by acidifying the iodine with an excess of the strongest nitric acid (1.5 s. g.) The liquid should be brought to dryness, and then boiled in a large glass retort (connected with a receiver) with an excess of strong nitric acid, the distilled products being constantly returned into the retort. By evaporating the residuary acid, iodic acid may be obtained in the dry residue. On dissolving it in water, adding starch and one or two drops of a solution of sulphurous acid, an intense blue colour is immediately produced, even when the iodic acid, and therefore the iodine, is only in infinitesimal traces. This process may be sometimes advantageously employed, when it is desirable to destroy the organic matter.

For an account of Iodide of Potassium, see chapter on Alkaline salts, ante, p. 230.

SULPHUR.

Sulphur is a metalloid which does not appear to possess any poisonous properties. In the state of hydrate (Precipitated Sulphur) it acts as a gentle laxative in large doses. There is not, so far as I am aware, any instance on record of its having produced injurious effects in the human subject. The only form of poisoning by sulphur which concerns the medical jurist, is that which relates to the Alkaline Sulphurets, for an account of which see ante, p. 232.

METALLIC IRRITANTS.

CHAPTER XXIII.

GENERAL CHARACTERS OF THE METALLIC IRRITANT POISONS. ARSENIC—PRECAUTIONS RESPECTING THE SALE OF THE POISON. ARSENIOUS ACID—TASTE—SOLUBILITY IN VARIOUS LIQUIDS—SYMPTOMS—CASES OF ACUTE POISONING—CHRONIC POISONING—ANOMALOUS CASES—ALLEGED ACCUMULATIVE PROPERTIES—EFFECTS OF EXTERNAL APPLICATION—POST-MORTEM APPEARANCES—PERIOD REQUIRED FOR INFLAMMATION AND ULCERATION—QUANTITY REQUIRED TO DESTROY LIFE—RECOVERY FROM LARGE DOSES—PERIOD AT WHICH DEATH TAKES PLACE—TREATMENT.

The irritant poisons, which are either salts or compounds of the true metals, are exceedingly numerous, and constantly demand the attention of medical practitioners. Several broad chemical distinctions may be drawn between them and the non-metallic irritants. They are all precipitated in neutral solutions by sulphuretted hydrogen gas, forming insoluble sulphurets of various colours. With the exception of arsenic, they are all precipitated by hydrosulphuret of ammonia; and with the exception of arsenic and its compounds, the vegetable salts of antimony and the chloride of gold, they are all precipitated by the ferrocyanide of potassium, forming ferrocyanides of various colours, (p. 130.) These tests, therefore, will serve in most cases to distinguish the metallic from the non-metallic irritants. A few of these bodies have a corrosive or chemical action on the viscera, as corrosive sublimate and nitrate of silver, but the greater number act by irritating and inflaming the alimentary canal. All are absorbed, and are liable to be carried off by the various secretions; but probably the chief source of elimination during the life of the person is the urine. In this secretion most of the metallic poisons, but especially arsenic and antimony, may commonly be found.

The metallic irritants do not admit of any easy subdivision, either according to differences in their mode of action or their chemical properties. The chemical differences are often as great among the salts of each metal as among the salts of different metals. They will therefore be here treated chiefly in the order of their practical importance, the compounds of those metals being taken first which most frequently give rise to medico-legal inquiries. We shall, in the first place, pass to the consideration of ARSENIC.

ARSENIC. ARSENIOUS ACID.

General Remarks.—The term WHITE ARSENIC is commonly applied to the arsenious acid of chemists. Arsenic acid is another compound which is highly poisonous, but has never, so far as I know, been used for the purposes of suicide or murder. YELLOW ARSENIC, or orpiment, is the sesquisulphuret of chemists. This is also poisonous, apparently because it contains a large portion of arsenious acid, which has not combined with sulphur. This often amounts to from fifteen to twenty per cent. of its weight. Orpiment has been, on several recent occasions, criminally used as a poison. White arsenic, or arsenious acid, is, however, that preparation which chiefly requires the attention of a medical jurist. In the years 1837-8, there were one hundred and eight-five cases of poisoning by this substance, the greater number of which were the result of suicide and murder (p. 156, ante.)

As a witness may sometimes have to infer *quantity* from the cost, it may be stated, that from half an ounce to one ounce of white arsenic is sold for twopence—one ounce and a half for threepence—if exceeding this, the charge is at the rate of one shilling a pound. This is stated on the authority of a highly respectable retail-house in London. Considering the great destruction of life which is continually taking place from arsenic, it is to be regretted that a substance so little required for any lawful purpose should be allowed to be sold in so free and open a manner. At almost any druggist's or grocer's shop throughout the kingdom, enough may be procured for a penny to destroy the lives of fifty persons! It is equally sold to men, women, and even children, upon the most absurd and trivial pretences; and the numerous murders and suicides annually committed by means of this poison, clearly prove that the precautions as to its sale, if observed at all, are futile and ineffectual. In the neighbourhood of London, a few years since, arsenic was sold by a druggist to a mere youth at a boarding-school; the boy took it and died. The druggist escaped, with the opinion of the jury, that his conduct was "culpable;" on the Continent, it is most probable he would have been heavily fined and imprisoned. The sale of alcohol and other fiscal articles is restricted, and there is no doubt that much crime might be checked, and many lives saved, by placing strong legal restrictions on the sale of arsenic and active poisons generally. It appears from the table (p. 156) that more than one-third of all the fatal cases of poisoning in England are occasioned by this substance. Druggists might to some extent prevent the criminal employment of arsenic by always selling the powder mixed with a few grains of powdered sulphate of copper and powdered ferrocyanide of potassium; about six grains of each to a drachm of arsenious acid. This would not interfere with its lawful use for poisoning vermin, but it would in many cases prevent the poison from being accidentally swallowed or criminally administered in liquids, because a red colour is immediately struck on the powder being wetted—a circumstance likely to attract particular attention. The humidity of the air, after a short time gives a rose-red tint to the arsenious acid by a partial production of the ferrocyanide of copper. Such a precaution could not prevent accidents in all cases, but I could point to many instances within my own knowledge where I have not the slightest doubt that it would have been the means of exciting suspicion and of saving life. This plan appears to me preferable to that of colouring the poison itself. It is important for the witness to remember, that arsenious acid is commonly sold in the country, and known to the ignorant, under the name of *Mercury.*

Taste of arsenious acid.—White arsenic is commonly seen under the form of a white powder, or in opake masses resembling enamel. It is called an acid from its power of combining with alkalies, but it possesses a very feeble acid reaction when dissolved in water. It is often described as having an *acrid taste*, but this does not appear to be correct; a small quantity of it has certainly no appreciable taste, a fact which may be established by direct experiment, and might be inferred from its very sparing solubility. It would appear from numerous cases on record, that it has beeen unconsciously taken in fatal quantities, in all descriptions of food, without exciting the least sensation on the tongue. Most of those persons who have been criminally or accidentally poisoned by arsenic, have not been aware of any taste in taking the poisoned substance;—it has certainly not been perceptible when the poison has been given in wine, milk, beer, and other simple liquids, or many lives would have been saved. With facts of this kind, it is extraordinary that Orfila should persist in making the unqualified statement that arsenious acid has "an acrid (*apre*,) not corrosive, but somewhat styptic taste, exciting salivation in a marked degree." (i. 377, ed. 4.) "*Sa saveur est âcre et corrosive.*" (i. 357, ed. 3.) This statement has had much influence on the medical evidence given in numerous cases of arsenical poisoning during the last half-century: for it has led to the vulgar belief that no person could take arsenic as a poison without being made fully aware of it by a strong and well-marked taste! There is no doubt that some of the after-effects of the poison, arising probably from its irritant action on the lining membrane of the fauces, have been frequently mistaken for the taste or impression produced by it on the tongue. In an accident by which a whole family was poisoned by arsenic mixed in a pudding, for the particulars of which I am indebted to Mr. Tubbs, of Upwell Isle, one person found the taste rather sweetish, rough and persistent, but experienced, at the same time, a burning sensation in the throat. Another felt his throat so hot that it was just as if he had swallowed pepper. In another case, which occurred to the same gentleman, who has paid great attention to the subject of arsenical poisoning, and has been very successful in its treatment, there was a better opportunity of judging of the taste of the poison, as no less than a teaspoonful (150 grains) of it was taken by an adult in the form of dry powder, without any fluid after it:—the man described the taste as "coarse and smartish." This is a fact somewhat in favour of the view entertained by Orfila: but it is nevertheless true that when dissolved,—when it is in small quantity, or mixed with common articles of food, the poison may be easily taken by a person quite unconsciously. As M. Flandin remarks, the differences of opinion on this subject are, perhaps, more apparent than real. He considers that arsenious acid has a decidedly hot taste only when it is taken in a very large dose, an experiment upon the performance of which no toxicologist is likely to venture; and that in small doses it is quite insipid. In a case of poisoning in which several persons partook of some meat on which powdered arsenic had been sprinkled, they stated that the meat had an unusual taste, something like that of a sour apple. (Des Poisons, i. 515.) In a very interesting case of arsenical poisoning (March, 1846,) for an accurate report of which I am indebted to Mr. J. H. Todd, coroner for Hants, the individual swallowed at least a drachm of arsenious acid, mixed up with half a tea-cupful of water, before breakfast, and on an empty stomach. These were circumstances highly favourable to the detection of the *taste* of the poison. After he had drunk off the dose, he said it was rather rough to the tongue; and for twenty minutes afterwards he complained that his tongue was very rough. In the case of the *Queen* v. *Maher* and *Lynam* (Kildare Lent Assizes, 1847,) some flummery was proved to have been poisoned with arsenic, and the taste of the flummery was complained of. My friend Dr. Geoghegan, who has communicated to me the particulars of this case, says that this taste could not be referred to the after-effects of the poison, and suggests that arsenic, like aconite and some other poisons, may have a taste after a certain interval. The facts here related will enable the medical jurist to form a judgment of the matter. If Orfila has given an exaggerated account of the taste of this poison, it seems clear that, in the state of powder and in large doses, it is in many cases capable of producing a decided and persistent impression on the tongue.

[According to the experiments of Navier, Devergie and those of Dr. Mitchell and Mr. Durand (*Phil. Jour. Pharm.* iv.) this poison in a saturated or hot solution, imparts a rough sensation to the tongue, somewhat similar to that caused by sulphate of zinc.—G.]

Arsenic not a corrosive.—Arsenic is not an irritant poison: it does not seem to possess any *corrosive* properties, *i. e.* has no chemical action on the animal tissues, and the changes met with in the alimentary canal of a person poisoned by it, are referrible to the effects of the inflammation excited by the poison, and not to any chemical action. I have not found that arsenic produces any effect on dead mucous membrane, like those poisons which are properly called corrosive. In an important case (*Waring's*,) tried at the Leicester Lent Assizes, 1842, the witnesses were closely pressed to say whether arsenic was or was not a corrosive poison; the deceased having been killed by arsenic in a few hours, and the local changes in the stomach being unusually well marked. They properly referred these changes to the violence of the inflammation, and not to the chemical action of the poison. It is the opinion of Dr. Christison, and of most English toxicologists, that arsenic is not a corrosive, nor does it appear, from its well-known chemical properties, how it should in any case operate as such. Nevertheless it is proper to state, that one instance at least is on record, in which it is alleged to have exerted a corrosive action. A man named *Soufflard*, on being condemned to death, swallowed three drachms of arsenious acid in powder: he vomited almost immediately. When seen shortly afterwards the lower lip was strongly cauterized (*fortement cautérisée*;) the mucous membrane was white, fissured, and the slightest touch produced excessive pain. The tongue was swollen, and the patient complained of a horrible taste in his mouth and fauces. After death which occurred in thirteen hours, the epithelium of the tongue was found destroyed. (Flandin, op. cit. i. 495.) Arsenic was detected in the stomach, the mucous coat of which was destroyed, or reduced to a gelatinous pulp; but it is not stated whether it was mixed with corrosive sublimate or any other poison. This action on the mouth is very similar to that produced by corrosive sublimate. According to the reporter of this case, arsenic in a large dose corrodes and destroys the tissues with which it comes in contact: in his opinion it acts like an acid or a caustic substance. (i. 557.)

Solubility of arsenic.—The *solubility* of this substance in liquids is a frequent question on trials. The action of water is materially influenced by circumstances. I have found by numerous experiments (Guy's Hospital Reports, iv. 81,) that hot water cooling from 212° on the poison in powder, dissolves about the 400th part of its weight. This is in the proportion of nearly one grain and a quarter of white arsenic to about one fluid-ounce of water. Water boiled for an hour on the poison and allowed to cool, holds dissolved the 40th part of its weight, or about twelve grains to one ounce. Cold water allowed to stand for many hours on the poison, does not dissolve more than from the 1000th to the 500th part of its weight; *i. e.* one-half grain to one grain of arsenic to nearly one fluid-ounce of water. The presence of organic matter in a liquid, renders the poison much less soluble. Thus, hot tea with milk and sugar, and cold porter, did not take up more than about half a grain to the ounce; while hot coffee and cold brandy did not dissolve more than one grain to the fluid-ounce. (Guy's Hosp. Rep. iv. 103.) Liquids, which are at all viscid or mucilaginous, may mechanically suspend the poison in almost any quantity, but in these cases it cannot be said to be dissolved. The solubility of arsenic was an important part of the evidence in the case of the *Queen* v. *Hunter*, tried at the Liverpool Lent Assizes, 1843. (See the medico-legal reports of this case which have been published, the one by Mr. Holland, and the other by Mr. Dyson of Manchester.) A medical witness must always take care to draw a distinction between the actual solution and the mechanical suspension of the poison in a viscid liquid, especially when it is necessary to determine whether the quantity taken was sufficient to kill. In reference to its solubility in cold water, I have found that when the quantity dissolved was not more than one-half grain to one ounce, *i. e.* less than the

1000th part, the solution had neither taste nor acid reaction whatever. The proportions are here stated in round numbers.

[There is much variance of opinion as to the solubility of arsenic. According to Klaproth and Bucholz, 1000 parts of temperate water take up two and a half parts, while the same quantity at 212° will dissolve 77.75 and retain 30 on cooling. Guibourt states that 1000 of temperate water will take up 9.6 of the transparent variety and 12.5 of the opake, whilst the same proportion of boiling water will dissolve 97 of the former retaining 18 on cooling, and 115 of the latter, retaining 29. The experiments of Dr. Mitchell and Mr. Durand show that the power of the solvent is influenced by the fineness of the powder, the time the fluid is in contact with it, and even the form of the vessel in which the solution is made. They state that 1000 of temperate water will dissolve 12 to 16 grains of either variety of the acid, whilst the same quantity of water at 212° will take up 148 and retain from 25 to 40 on cooling, according to the time the boiling fluid is in contact with the arsenic.—G.]

A medical witness is often asked the weight of common or familiar measures of arsenic in powder. It may therefore be stated that, from experiment, I have found a teaspoonful of powdered arsenic to weigh 150 grains,—a tablespoonful to weigh 530 grains,—and a pinch, or the quantity taken up between the finger and thumb of an adult, to weigh 17 grains. These weights are here given as the results of actual experiment: but they are of course liable to vary. The exact weight of a fluid-ounce of water is also subject to great variation in the measures used by druggists. The solubility of arsenious acid is said to be increased by admixture with nitre, and its action is described as being then more energetic. (Med. Times, Dec. 1844.) Arsenic is soluble in most organic liquids, as milk, coffee, tea, wine, brandy, and even oil. Although it is less soluble in these liquids than in distilled water, it is nevertheless, taken up in sufficient quantity to occasion serious accidents, and even to destroy life. In 1845, a man was found guilty, at Madrid, of putting arsenic into oil with the intent to destroy the life of his parents. Arsenic was found in the oil, and a portion of it given to a dog, caused its death, (Journal de Chimie, 1845, p. 651.) This poison is very soluble in strong muriatic acid; and any alkali or alkaline carbonate dissolved in a liquid, greatly increases its solubility.

SYMPTOMS.

These will vary according to the form and dose in which the poison has been administered. The *time* at which they come on is generally in from half an hour to an hour after the poison has been swallowed. This is the average period. I have known them to appear in a quarter of an hour. Dr. Christison mentions one instance in which the symptoms began in eight minutes; but in the case of *Lofthouse*, tried at the York Lent Assizes, 1835, the symptoms were proved to have attacked the deceased while he was in the act of eating the cake in which the poison was administered. On the other hand, in an instance communicated to me by Mr. Todd, where one drachm had been taken on an empty stomach, no symptoms appeared for two hours; in one reported by Orfila, the symptoms did not show themselves for five hours; and in another which occurred to Dr. Lachèse, where a large dose was taken, the symptoms did not appear for seven hours. (Ann. d'Hyg. 1837, i. 344.) There may be every variety between these extremes. A case will be presently mentioned, in which their appearance was protracted for *ten* hours—the maximum period yet known. (Post, p. 317.) A very remarkable instance occurred to M. Tonnelier, in which the poison was taken by a young female at eleven o'clock in the morning, and no well-marked symptoms occurred for *eight hours*: there was then violent vomiting. After death a cyst, formed of mucous membrane, and containing arsenic, was found in the stomach; the poison having thus become sheathed over! (Flandin, i. 535.) (Case of *Reg.* v. *Foster*, Bury Lent Assizes, 1847.) In all cases in which arsenic enters the system from without, as by its application to the skin and to ulcerated or diseased surfaces, the symptoms are rarely manifested until after the lapse of several hours. In the opinion of Dr. Christison, the symptoms may be retarded by sleep.

Their nature.—The individual first experiences faintness, depression, nausea, and sickness, with an intense burning pain in the region of the stomach, increased by pressure. The pain in the abdomen becomes more and more severe; and there is violent vomiting of a brown turbid matter, mixed with mucus, and sometimes streaked with blood. These symptoms are followed by diarrhœa, which is more or less violent; and this is accompanied by severe cramps in the calves of the legs. The matters discharged from the stomach and bowels have had in some instances a yellowish colour, as it was supposed, from a partial conversion of the poison to sulphuret The vomiting is in general violent and incessant, and excited by any substance taken into the stomach. There is tenesmus, and the alvine evacuations are frequently tinged with blood. There is a sense of constriction, with a feeling of burning heat in the throat, often accompanied by intense thirst. The pulse is small, very frequent, and irregular; sometimes wholly imperceptible. The skin is cold and clammy in the stage of collapse; at other times it is very hot. The respiration is painful from the tender state of the abdominal parietes. Before death, coma sometimes supervenes, with paralysis, tetanic convulsions, or spasms in the muscles of the extremities. In one instance trismus appeared in three quarters of an hour. (Orfila, i. 449.) Such is the ordinary character of the symptoms in an acute case of arsenical poisoning, *i. e*, where from half an ounce to an ounce of the poison has been taken.

Chronic poisoning.—Should the person recover from the first effects, and the case be protracted, or should the dose have been small and frequently administered, there will be inflammation of the conjunctivæ, with suffusion of the eyes, and intolerance of light,—a condition which is, however, often present with the early symptoms above described. There is also irritation of the skin, accompanied by an eruption, which has been called eczema arsenicale; local paralysis and other symptoms of nervous disorder, are very common sequlæ. Exfoliation of the cuticle and skin of the tongue, with the falling off of the hair, has likewise been witnessed. (Case of the *Turners*, 1815, Marshall, 119.) Salivation has been observed to follow, especially when small doses of the poison have been given for a length of time. (Med. Gaz. xvi. 790.) Strangury and jaundice have also been noticed among the secondary symptoms. (Marshall on Arsenic, 44, 111.) A well-marked case of *slow poisoning* by arsenic is recorded by Flandin. It resembles in some respects the case of *Blandy* (ante, p. 98,) except that it did not terminate fatally. As this mode of *secret* poisoning appears to be on the increase, it may be as well to state the facts. A woman put daily into the soup of her fellow-servant, a very small quantity of arsenious acid in powder. Shortly after dinner this person was seized with vomiting, which led to the rejection of the food and poison before the latter had caused any serious mischief. As this practice was continued for about six weeks, the stomach became exceedingly irritable, there was pain in the bowels, and the woman became much emaciated. There was also spitting of blood, with such a degree of nervous irritability, that a current of air caused an attack of spasms and convulsions. When the patient found that she could not bear any thing on her stomach, she left the place and passed two months in the country, Her health became gradually re-established there, and she returned to resume her usual occupations. The prisoner, however, renewed her attempts; and to make sure of destroying life, gave her one morning in coffee, a strong dose of arsenious acid in powder: violent vomiting ensued, and the poison was expelled with the breakfast. Arsenic was detected in the vomited matter, and the explanation of the cause of the long previous illness then became clear. Under proper treatment the patient recovered. (Op. cit. i. 510.) I believe this mode of poisoning to be more frequent in this country than is commonly supposed; and it behoves practitioners to be exceedingly guarded in their diagnosis, for the usual characters of arsenical poisoning are completely masked. The symptoms might be easily referred to chronic inflammation, or ulceration of the stomach leading to perforation. I have lately had to examine a case of this kind, where the death of a person had been caused by his housekeeper under somewhat similar circumstances. The crime was not discovered until after the lapse of two years; and from the small dose given, and repeated vomiting during life, no arsenic could be detected in the body.

In a case communicated by Mr. Jones to the Provincial Journal, (Nov. 18, 1843, p. 127,) the action of small doses of arsenic, administered medicinally, produced the well-marked effects of *slow* poisoning. The liquor arsenicalis was prescribed for a lady affected with cutaneous disease, in doses of five drops (=1-24th of a grain) of arsenious acid, and fifteen drops (= 1-8th grain,) three times a day. After continuing these doses more or less for a month, she was found labouring under the following symptoms: obstinate diarrhœa, frequent griping pains in the bowels, with almost constant desire to act; considerable tenderness over the whole abdomen, which was distended; constant pain and nausea after taking food, and frequent vomiting; skin cool and dry; intense thirst; tongue clean and red, resembling raw beef; pulse 100, small and feeble; sense of constriction in the throat, and copious flow of saliva; she had some gastric cough, with frequent raking of the throat and fauces, and expectoration of a muco-purulent secretion mixed with blood. There was much pain and tenderness down the spine, with frequent muscular tremors; and a crampy feeling of the lower extremities, with partial loss of motion and sensation; they were swollen, of a livid colour in places, and showed a tendency to slough. There was great emaciation; want of sleep, owing to the irritable state of the bowels; motions white, watery, and frothy; urine scanty, high coloured, and passed with an effort. From this account of the symptoms it is obvious that a case of slow poisoning by arsenic might very easily be mistaken for gastro-enteritis, and treated accordingly. Dr. Pfeufer has recently published an interesting case of this masked form of arsenical poisoning, in which there was general and well-marked paralysis of the whole of the muscular system, and a complete loss of sensation in the fingers. It was only after a year that the patient began to recover the use of his extremities. It appeared that his wife had been in the habit of giving him small doses of arsenic in his food. None of the articles of food, or of the matters vomited, could be procured for analysis; nevertheless, the evidence of chronic poisoning from symptoms was considered to be sufficiently strong to justify a conviction. (Zeitschrift für Rationelle Medizin, B. vi. H. i. 102, Heidelberg, 1847.)

Anomalous cases.—The symptoms described under the *acute* form of poisoning may not be met with in every instance. Thus the *pain*, which is usually excruciating, like a fire burning within the body, is sometimes absent. In a well-marked case of poisoning, which occurred in October 1839, a dose of from one ounce to two ounces of arsenic was taken; there was no pain except of the most trifling character just before death. It has been supposed that this symptom was absent when the dose was large; but a case occurred in Guy's Hospital, in 1836, where only forty grains had been taken, and the patient died without complaining of pain. (Guy's Hosp. Rep. iv. 68.) There are many similar instances on record. The symptoms of alvine irritation are seldom wanting, or there is vomiting, if there should be no purging. In one case of criminal poisoning by arsenic, in which I was consulted by Mr. Veasy, which was tried at the Bedford Spring Assizes, in 1842, there was neither vomiting nor purging. The quantity of poison taken must have been very small. In a case which occurred to Dr. Feital, although half an ounce of arsenious acid had been taken, there was no vomiting, (ante, p. 86.) Great *thirst* is a common symptom, but this is sometimes absent. With respect to the urinary secretion there is no certain rule: it is sometimes suppressed, as in several cases reported by M. Flandin; at other times it is natural, or only slightly diminished. (Des Poisons, i. 521.) It is necessary for a medical jurist to attend to these anomalies, as otherwise the symptoms of arsenical poisoning may be easily mistaken for those of disease.

The following case, quoted by Belloc, is in this point of view remarkable. A young woman, with the design of committing suicide, procured a lump of arsenic. She began by biting it; but as she could not procure sufficient in this way, she broke it up into coarse fragments, put them into a glass of water, and swallowed them. This was in the morning, and she went the whole of the day without suffering any marked uneasiness. At six o'clock in the evening she was seen by M. Laborde,—and there were then no febrile symptoms. At eight o'clock she suffered from pain in the abdomen. At eleven o'clock she appeared to be more calm than ever, and had a strong desire to sleep. At three in the morning she sat up in her bed, complained a little of her stomach, and then died, without the least appearance of suffering. She vomited some fragments of arsenic before death. On opening the stomach, the vessels were found gorged, and there were coagula of blood in the folds of the mucous membrane at the cardia. There were marks of excoriation about the lips, mouth, and œsophagus. (Cours de Méd. Lég. 122.) This is in every point of view a most remarkable case. No one acquainted with the usual effects of arsenic could have suspected it to be a case of arsenical poisoning. It therefore most strikingly shows, as Belloc observes, the absolute necessity for post-mortem inspections in all unexplained cases of death. The symptoms were probably more protracted in their appearance than in any other instance yet recorded, not having manifested themselves, as we may infer, until *ten hours* after the taking of the poison. They appear to have been at no time severe—there is no account of diarrhœa having been among them—they underwent a complete remission before death, and the deceased expired as from the effects of a narcotic, about seventeen hours after taking the poison.

It might be supposed, *à priori*, that the symptoms of irritation occasioned by arsenic, would be protracted in their appearance or mitigated in their character when the poison was taken mixed with opium: but in one well marked case in which half a teacupful of arsenic was swallowed with upwards of an ounce of laudanum, there was severe pain, abundant vomiting for two hours, and death took place in six hours (Med.-Chir. Rev. vii. 170.)

Is arsenic an accumulative poison?—It is well known that arsenic is carried into the circulation by absorption, and the question here proposed depends on whether its elimination in the living body takes place with the same rapidity as its absorption. I am not aware of any facts which show that arsenic can be taken in non-fatal doses for a certain period, accumulate in the body, and then suddenly give rise, without increase of dose, to all the marked symptoms of poisoning. This important medico-legal question arose in the case of *Lacoste*. From the presence of arsenic in the remains of the body, and from the symptoms under which deceased died, it was alleged that his death had been caused by poison. The quantity of arsenic found in the liver was about the 1-13th of a grain, and a still smaller proportion in the other viscera. In defence, it was alleged that deceased was in the habit of taking Fowler's solution (of arsenic) daily as a remedy for a skin disease under which he was labouring. The Procureur-général admitted this, but contended that, as the man was in the habit of taking arsenic medicinally, it was very improbable that he should have taken an overdose (a fatal dose:) and it could not therefore, be supposed that he had died through his own imprudence. The president inquired whether innocent doses of arsenic could possibly accumulate in the body and suddenly occasion symptoms and death. M. Devergie replied in the negative, M. Flandin in the affirmative—the latter resting his opinion not upon any facts observed in the human body, but upon the assumption that the elimination of the poison might not always go on *pari passu* with its absorption. (Flandin, Des Poisons, i. 484.) The opinion then given, was based upon experiments on animals. M. Flandin states that he has confirmed it by observations on the human subject made since the trial. The facts, however, are not stated, but he quotes the opinion of an English writer that Fowler's solution should not be long continued; because arsenic, like lead, might accumulate in the system and ultimately produce paralysis. The woman Lacoste was acquitted of the murder: it was considered that the small quantity of arsenic found in the body might possibly have been derived from that which had been used medicinally. This question of *accumulation* must therefore be regarded as still undetermined: at the same time, there are no facts which render it probable that arsenic is an accumulative poison; *i. e.* that it can collect in the system, without symptoms, and suddenly produce those effects observed in the administration of a fatal dose. The case of slow poisoning related at p. 257, is decidedly adverse to the view that arsenic is an accumulative poison. M. Flandin quotes the case, but does not perceive that it is directly opposed to the inference which he has drawn. In a subsequent part of his work he also relates some experiments, the results of which show that arsenic has no accumulative properties. M. Danger and himself gave to animals, gradually increasing doses of arsenic in powder, mixed with their food. In beginning with the 1-65th part of a grain they found, after an interval of nine months, that the animals might be made to take upwards of *fifteen grains* of arsenious acid in twenty-four hours without any influence on their appetite or health. During this time, their urine was frequently analysed at different intervals without any arsenic being detected. Three days after the last dose of arsenious acid was given, the animals were killed, and their organs successively analysed, but no arsenic was detected in the viscera, flesh, or bones. (i. 737.) M. Flandin thus appears to have conclusively proved, contrary to his own opinion, that arsenic certainly does *not* accumulate within the body!

From this account of the symptoms produced by arsenic, it will be seen, that there is great difficulty in classifying cases. On referring to many which I have been enabled to collect, the symptoms do not appear to bear any relation to the *quantity* of poison or to the *form* in which it is administered. Those indicative of irritation in the alimentary canal, or of coma, have equally occurred, whether the dose was small or large, and whether the poison was in the state of powder or solution. The same observation may be made of other cases in which symptoms of an affection of the brain have followed those of irritation. Hence it appears to me that any useful generalization is impossible, for the exceptions are so numerous as to show that the arbitrary divisions which must necessarily be made, can be of no practical utility.

The following case, which occurred to Dr. May, shows an entire absence of the most prominent symptoms of arsenical poisoning. A child, aged twenty months, ate some paste consisting of honey, flour, and arsenic, prepared for destroying mice. He was caught in the act of eating it, and a considerable portion was forced from his mouth. Some ipecacuanha wine was given to him, and he vomited freely, the ejected matter consisting chiefly of mucus of a yellowish colour, with some of the paste suspended in it. The vomiting was encouraged, and milk was freely given. Between the fits of vomiting he appeared lively. In about two hours he had two natural motions, and was sleeping calmly as usual: he had had no pain; respiration a little hurried. In about six or seven hours he became somewhat restless, but there was no expression of pain. Soon after this, he became worse, the surface was cold, lips livid, eyes sunk, the pupils fixed and rather dilated, pulse scarcely perceptible, respiration feeble, accompanied with sighing. After lying for half an hour in this condition, he expired without a struggle, nearly eight hours subsequent to the accident. On inspection, the stomach and intestines exhibited little deviation from the healthy state. The stomach contained mucus and a portion of the paste, but the villous surface presented no signs of inflammation. (Prov. Med. Journ., July 16, 1845, 453.) Dr. May very properly observes that had not the child been seen to eat the paste, there was not a symptom, nor after death any morbid appearance, to indicate the true cause of the child's illness.

It has been supposed that stupor and other symptoms of cerebral disturbance were more likely to occur when the dose of arsenic was large; but a case was communicated to the London Medical Review (April 1811, p. 188,) by Mr. Soden, of Coventry, which shows that, with a large dose of arsenic and rapid death, there may be violent symptoms of gastric irritation, and few or no symptoms indicative of nervous disorder. A man, aged twenty-two, purchased seven ounces of finely powdered arsenic, and swallowed, between seven and eight in the morning, not less than four and probably six ounces of the poison. In about half an hour he was found vomiting: there was severe pain in the abdomen, rapid pulse, and slight convulsions of the legs. In two hours diarrhœa supervened, and there was constant inclination to pass urine: the pain in the bowels became almost intolerable, the convulsive motions of the limbs more frequent, and the pulse more feeble, but still very quick. According to the antidotal doctrines at that time prevalent, sulphuret of potash (potassium) was largely exhibited to him. He died in less than four hours, after a dreadful fit of convulsive laughter, his limbs becoming suddenly rigid. In this case, there was neither stupor nor faintness, but there was severe pain, with convulsions. On inspection, the stomach was found highly inflamed, "the mucous coat looked as though it had been beautifully injected," and two ounces of arsenic were found in the cavity of this organ.

The following is a still more recent instance, which was referred to me for examination by Mr. Carter of Newbury, in July 1845. A female, aet. 22, swallowed a large dose of arsenic. She was immediately afterwards seized with intense thirst, severe burning pain, violent vomiting, and diarrhœa continuing incessantly until death, which took place in *seven* hours. There were no nervous symptoms. The stomach and the whole of the alimentary canal were extensively inflamed. Arsenious acid was detected in large quantity, mixed with flakes of mucus, in the stomach, throughout the small intestines and in the cæcum. (G. H. R. vol. iv. Oct. 1846, 458.) On the other hand, if severe symptoms of alvine irritation thus follow large doses, those indicative of an affection of the nervous system may be produced by comparatively small doses of the poison. I am indebted to Mr. Todd, coroner for Hants, for the particulars of the following interesting case. A man, aet. 24, swallowed about six o'clock in the morning on an empty stomach, a drachm of white arsenic mixed with half a tea-cupful of water. He complained of the mixture having a rough taste, but he went to his work, and it was only the suspicion that he had made a mistake and swallowed poison, that induced him to go to the house of a surgeon, about two hours afterwards, for an emetic. He then appeared to be very well, and made no complaint; the surgeon could not detect about him any symptom of arsenical poisoning, and was inclined to disbelieve the man's statement. Vomiting was produced by sulphate of zinc, and he threw up some yellow-coloured matter. He saw the man again in five hours: he was then sitting in a drowsy state, with a countenance expressive of great anxiety, a blue tinge on the hands, pulse very feeble, and there was profuse diarrhœa. He complained of *no pain*, and said that he wanted to go to sleep. Vomiting had ceased. He died in this calm state in the evening, about fourteen hours after he had taken the poison.

The mixed nature of these cases will also be obvious from the following, reported by Dr. Letheby. A girl, aged nineteen, swallowed two grains and a half of arsenic dissolved. Restlessness during the night, and slight pain, were the only symptoms experienced. In the morning she became sick, complained of great thirst, and the pain was more intense. Vomiting increased, followed by diarrhœa, the countenance was pinched, and the extremities became cold. From this state she rallied, and slept comfortably the following night. The next morning she became worse, being cold and drowsy, the pulse scarcely perceptible, and she was passing in to a state of incipient coma. In this condition she sank about thirty-six hours after taking the poison. The stomach was pale and nearly empty, and its mucous coat was raised by a number of vesicles containing air. (Med. Gaz. xxxix. 116.)

Mr. Foster of Huntingdon has kindly communicated to me three cases of poisoning by arsenic of great interest, not merely with regard to the variety in the symptoms, but also in the post-mortem appearances, and the rapidity of death.

E. B., the mother of five children, æt. 37, had been in a desponding state of mind, with general debility of the system, for some weeks. On the 26th of May, 1638, Mr. Foster was hastily summoned to her residence, a distance of eight miles, and arrived about one o'clock, P. M. He found that a medical friend had been in attendance for an hour and a half. The woman had administered arsenic to her two children, and had then taken it herself. There were no means of ascertaining the quantity administered; she had mixed it, for herself and eldest child, in the yolk of an egg, and for the infant in pap. From the evidence of the servant-girl, it appears that the poison must have been taken some time between half-past nine and eleven o'clock, A. M. The difference in the effects produced on each patient was remarkable, and requires separate consideration.

Case I.—The mother, when first seen, shortly after eleven o'clock, was sensible, and confessed to having given the children arsenic, and taken it herself. She was vomiting violently, and complained of a burning sensation at the pit of the stomach; there was constant tenesmus, with dejection of mucous stools; pulse small and rapid; violent delirium soon followed, with tetanic convulsions, requiring the utmost exertion of four persons to hold her in bed; the conjunctiva of the eye became intensely injected; the pupils, from being minutely contracted, became exceedingly dilated: the eyeballs fixed upwards; the mouth drawn in all directions. This state continued for some minutes, when calmness and repose followed, during which she expired, about one o'clock, not more than three hours and a half after having taken the poison.

Inspection.—The stomach, small intestines, and bladder, on their peritoneal surface, exhibited much inflammatory vascularity. The stomach contained a pint of thin, glairy fluid of a yellowish colour; some white powder (proved to to be arsenic) was seen to adhere in some places to the villous coat, which in every part was red and inflamed, but not nearly so much as in the children. The heart was healthy; much dark blood on the right side. The lungs were healthy, except their apices, which were studded with tubercles in the first stage, and where there were some hard, dark-coloured patches of an irregular surface, and of the size of a sixpence, similar to the cicatrices of cured tubercles, as mentioned by Carswell, Andral, and Louis. The other parts examined afforded no evidence of disease.

Case II.—Elder child, (girl,) aged two years and a half, when first seen was in a comatose state. She had been sick and convulsed, and had suffered severe pain, but now there was complete insensibility; the face was swollen and of a livid hue; the pupils dilated; the breathing difficult; the extremities cold. She was, in fact, in a complete state of narcotism, and died about half-past eleven, not more (probably less) than two hours after taking the dose.

Inspection.—The stomach contained three or four ounces of fluid, with some particles of white powder; the redness of the villous coat was of a vermilion hue, much more so than in Case I.; but much less than in the infant, Case III. The other appearances were similar to those in the mother.

Case III.—The infant, aged five months, was found, about three hours after swallowing the poison, in great agony, with severe bilious vomiting and convulsions, the extremities cold, the lower ones retracted to the abdomen, which was swollen and tense, the countenance pale, and pulse imperceptible. The usual remedies were administered, but the child gradually sank, and died about four o'clock, six hours and a half after taking the arsenic.

Inspection.—The appearances were the same as in the foregoing cases, except that the villous coat of the stomach was in the highest state of inflammation, and in the greater part of its extent the redness was of the brightest scarlet colour; there was an effusion of dark blood in patches; no ulceration; no thinning of the coats. An analysis of the contents of the stomach in each case afforded the usual colours and deposits with ammonio-sulphate of copper and ammonio-nitrate of silver; and arsenic in its metallic state was procured from the contents of each stomach.

It will be observed, that in Case II. (of the little girl) death followed in not more, probably in less, than *two* hours. As this was a fact of some importance, Mr. Foster made minute and careful inquiries. The child died at half-past eleven; the husband and servant both stated that they were present with the poor woman during the morning until half-past nine (they breakfasted with her as usual at nine,) and during this time it was scarcely possible for her to have taken the arsenic without their knowledge. At half-past nine the husband left to go into the fields; the servant went into the kitchen; the mother almost immediately proceeded up stairs with two of her children, and there remained until they were discovered in the state above mentioned. The supposition that this was the time the arsenic was taken, is further confirmed by the fact, that the mother earnestly entreated that an elder child might stay from school that morning, she having intended, as she stated before her death, to give the child some of the arsenic as well as the other children. The mother died in three hours and a half, whilst the infant lived six hours and a half. It will also be remarked, that there was considerable variety in the symptoms and pathological appearances in each case:—Case I. mother: intense excitement of the nervous system, with comparatively slight local inflammation of the stomach. Case II. elder child: disturbed action of the heart and circulating system, almost amounting to narcotism, with a greater degree of inflammation of the stomach. Case III. infant: very great and violent local inflammation, with slighter remote symptoms. In all three cases death was unusually rapid.

Hitherto, the direct effects of arsenic on the alimentary canal, and its remote effects on the brain and nervous system, have been considered. Some toxicologists affirm, that the poison has a specific action on the *kidneys*, and that it causes suppression of urine. MM. Danger and Flandin have advocated this view; while M. Orfila contends that the urinary secretion is never suppressed in arsenical poisoning. It is quite unnecessary to examine the arguments which have been adduced in support of these conflicting views—the fact being, as those who have attended to the subject of arsenical poisoning, irrespective of theory, well know, that sometimes there is strangury or suppression of urine, while in other instances the secretion goes on as usual, (see p. 258.) The remote influence of arsenic upon the *heart* is proved by the faintness and syncope which are so frequently met with in the progress of the case.

Effects of external application.—Arsenious acid, it is well known, when applied to wounded or ulcerated surfaces, becomes absorbed, and produces the usual effects of poisoning. A case is reported in Rust's Magazin, where a man covered his head with arsenic in powder to act as a depilatory. He was affected with the usual symptoms of arsenical poisoning, excepting diarrhœa, and he died on the *twentieth* day. The interior of the stomach, as well as the lower part of the œsophagus, was generally inflamed.

The following case, communicated to me by Mr. Tubbs, proves that arsenious acid when rubbed on the skin, has decidedly a local *irritant* action. A man, who was subject to piles, was in the habit of anointing himself with lard. By mistake, on one occasion, be used some white ointment containing arsenic. The next day he complained of an intolerable itching of the anus and scrotum; and, on examination, the parts were covered with pustules surrounded with an inflamed base. They resembled those which are produced by arsenic. On examining the matter from the pustules, it was found to contain arsenious acid. Frictions of lime-water and oil were used, and the patient soon recovered.

Instances of arsenic thus destroying life when applied externally, are by no means unfrequent. Two cases of its operating fatally in children, when applied to the skin of the head for tinea capitis, will be found in the Annales d'Hygiène, 1830, ii. 437. In both, the mucous membrane of the stomach was found inflamed, and in one extensively. A trial has recently taken place (*Reg.* v. *Port*, Chester Winter Assizes, 1844,) in which a man, pretending to cure cancer, was charged with the death of a female, by the application of an arsenical plaster, as it was supposed, to the breast. The woman died in a fortnight. No satisfactory evidence was obtained of the symptoms during life, except that there had been vomiting; and the accused had taken care to remove the plasters so soon as serious symptoms began to appear,—hence there was no direct chemical evidence of the nature of the substance actually employed. This case, however, shows the great utility of the discovery of the absorption of arsenic into the body. Dr. Brett, of Liverpool, was able to detect the absorbed arsenic in the substance of the stomach, liver, and spleen: the quantity detected was less than a quarter of a grain. The œsophagus, stomach, and intestines were found extensively inflamed. Notwithstanding this evidence, which appears to have been particularly clear, the prisoner was acquitted! The learned judge, in charging the jury, observed, "that the quantity of arsenic was exceedingly small" (see ante, p. 117,) although why *any* arsenic should be found in the stomach, liver, and spleen of a human being simultaneously with extensive inflammation of the stomach and bowels after the application of a plaster (not produced) to a diseased breast, is entirely unexplained! We can only suppose, admitting the verdict to be correct, that the arsenic found, was spontaneously generated by some mysterious process in the tissues of the body, and that the inflammation of the alimentary canal was a mere coincidence! In January 1845, a man in this city died apparently from the effects of arsenic absorbed through the skin of the arm. He was engaged in the manufacture of candles, to which arsenic was added in large proportion, and it was supposed that an abrasion of the skin had facilitated the absorption of the poison. The medical opinion given at the inquest, was decidedly that the deceased had died from the effects of arsenic thus introduced into the system. M. Flandin states, that on one occasion he had to examine the viscera of a woman who had been killed by the application of an arsenical powder for the cure of a scirrhous breast. The arsenic (absorbed) was discovered in various parts of the body, but especially in the liver, which contained as much as is usually found when the poison has been swallowed. (Des Poisons, i. 230):—the quantity was greater than that found in all the other organs together. This case presents many points of interest. The poison did not begin to produce its well-marked effects until after the lapse of about *ten hours*. Death took place in about six days, and the urine was suppressed throughout. The mucous membrane of the stomach and intestines was in its natural state: in the duodenum it was slightly swollen or thickened. (Flandin, i. 502.)

The powder used by quacks as an application to scirrhous breasts is commonly a compound of arsenious acid, realgar, and oxide of iron. In this instance, it was formed of 75 parts of the first-mentioned substance, and 25 parts of the two last. The quack stated that he did not apply more than four or five grains.

The symptoms are slower in appearing on these occasions that when the arsenic is swallowed, probably from the diseased surface being less absorbent than the mucous membrane of the stomach; but in an experiment performed by Mr. Swan, in which arsenious acid was introduced into a wound on the back of a dog, vomiting came on in two hours, and the animal died in six hours. The villous coat of the stomach and of the intestines was found inflamed. (Action of Mercury, p. 33, 1847.) In a case somewhat similar to that above related, M. Flandin discovered arsenic in the breast to which the substance had been applied, but in no other part of the body. (Op. cit. i. 550.) Some interesting facts regarding this form of poisoning will be found in the Ann. d'Hyg., 1846, ii. 131.

This mode of destroying life by the local application of arsenic to ulcerated surfaces, has never, so far as I am aware, been resorted to by criminals. It is obvious, that when death is not a consequence, serious injury to health might ensue—life might be endangered,—and to those who have framed our criminal statutes it ought to be a subject of consideration, whether this offence could be strictly comprised within the statute against poisoning. (1 Vic. lxxxv. Sec. 2—see page 19.) Would the words "administer or cause to be taken" be considered to include the *external* application of arsenic criminally? If this be a subject of doubt, the punishment of a serious offence is not provided for in our present law. The external application of orpiment in the form of ointment has also caused death. (See SULPHURETS OF ARSENIC, post.) Belloc states that he employed arsenical compounds externally to scirrhous tumors of the eye, cheek, and nose, without any serious consequences resulting. (Cours de Méd. Lég. 121.)

Several cases are reported in which arsenic has acted as a poison through the *unbroken skin*. Some of them are of old standing, and do not appear to have been very accurately observed. (Flandin, i. 542.) If the arsenic be in solution, it may become speedily absorbed: but when in powder, absorption would take place much more slowly. It is well known that comparatively insoluble substances may be introduced into the system by the endermic method, and arsenic does not appear to present any exception to this mode of operation. The thin skin of the human subject appears to absorb the poison more readily than the hard thick skin of animals; but M. Flandin found that dogs were speedily killed when arsenical ointments were rubbed upon the skin of the abdomen, or on the inside of the thighs. (Op. cit. i. 544.) All the symptoms of arsenical poisoning, although not appearing for two or three days, have been witnessed in the human subject in those cases in which powdered arsenic has been used as a depilatory.

[Several cases have been reported in our Medical Journals, where death has ensued from the external application of preparations containing arsenic, a synopsis of which has been given by Dr. Lee in his valuable notes to Guy's Forensic Medicine.—G.]

POST-MORTEM APPEARANCES.

The striking changes produced by arsenic are generally confined to the stomach and intestines. They are commonly well-marked in proportion to the largeness of the dose and the length of time which the individual has survived after taking the poison. Our attention must first be directed to the *stomach*. Arsenic seems to have a specific effect on this organ; for, however the poison may have entered into the system, whether through a wounded or ulcerated surface, or by the act of deglutition, the stomach has been found inflamed. Inflammation of this organ cannot, then, be always considered to depend on the local irritant action of the poison on the stomach.

The mucous membrane of the stomach, which is often covered with a layer of mucus, mixed with blood, and with scattered white pasty-looking patches of arsenious acid, is commonly found red and inflamed; the colour which is sometimes of a dull or brownish-red, becomes brighter on exposure to the air; at other times it is of a deep crimson hue, interspersed with black-looking striæ of altered blood. The redness is usually most strongly marked at the greater extremity; in one case it may be found spread over the whole mucous surface, giving to it the appearance of red velvet,—in another it will be chiefly seen on the prominence of the rugæ. Blood of a dark colour is effused in various parts between the rugæ, or beneath the lining membrane, an appearance which has been mistaken for gangrene. The stomach often contains a mucous liquid of a dark colour tinged with blood. The coats are sometimes thickened in patches, being raised up into a sort of fungous-like tumour, with arsenic imbedded in them: at other times they have been found thinned. The mucous membrane is rarely ulcerated, and still more rarely gangrenous. Perforation of the coats is so uncommon a result of arsenical poisoning, that there are only three instances on record. The mucous glands of the stomach have been found enlarged; but this is by no means an unusual morbid appearance from any cause of local irritation, without reference to poisoning. Various morbid appearances are said to have been met with in the lungs, heart, brain, and urinary organs; but they do not appear to be characteristic of arsenical poisoning. It is undoubtedly to the stomach and intestines, that a medical jurist must look for the basis of medical evidence in regard to post-mortem appearances.

[Dr. Storer (Bost. Med. and Surg. Journ. xxiii. 345,) is of opinion that the most characteristic mark of poisoning by arsenic, isecchymos is of the mucous membrane, and a bloody tinge of the fluids in the cavity of the peritoneum and pericardium.—G.]

Period required for inflammation.—A witness is often asked in a Court of law how long a time is required after the taking of the poison, for the production of these well-marked appearances in the stomach, more especially of *inflammation* of the mucous membrane. In reference to this question, we have the following facts. In a case which I had to examine, a large dose of arsenic had been taken;—the man, aged 21, died in five hours, and the stomach was found intensely inflamed, especially about the greater curvature. In a case that occurred to Mr. Thompson of Nottingham, half an ounce of the poison was taken; the patient died in six hours, and the stomach was found uniformly red and inflamed. In another that occurred to Dr. Booth of Birmingham, the same quantity of arsenic was taken; the patient died in six hours and a half: on inspection, the œsophagus was inflamed, the whole internal surface of the stomach was of an intense scarlet colour, and there was redness and increased vascularity of the duodenum, jejunum and ileum. In *Waring's* case already referred to, where but a small quantity of arsenic could have been taken, the whole of the stomach and intestinal canal was found highly inflamed, although the deceased could not have survived four hours. In Mr. Foster's cases, death occurred in one, a child, at the end of *two hours:* in the second an adult, at the end of *three hours and a half;* and in the third, after the lapse of about six hours. (Ante, p. 262.) In each of these, the stomach was found highly inflamed, and in the one that proved fatal in two hours, the mucous membrane had a vermilion hue. This last I believe to be the shortest period at which inflammation of the stomach from the effects of arsenic, has been met with.

Period required for ulceration.—Another question put to a witness may be this,—What period of time is required for *ulceration* of the mucous membrane to take place, as an effect of this poison? If arsenic has destroyed life with unusual rapidity, and the stomach is found ulcerated, an attempt may be made to refer this ulceration to some other cause. Such an attempt was made in the case of *Rhymes,* which was the subject of a criminal trial in 1841. (Guy's Hospital Reports, Oct. 1841, p. 283.) I found ulceration of the mucous membrane, which had been completely removed in patches, although the deceased survived the effects of the poison only *ten* hours. The deposition of the arsenic in and around the ulcers, as well as the appearance of recent inflammation about them, left no doubt that they had been produced by the poison, and were not owing to previous disease, as it was attempted to be urged in defence. When no arsenic is found in the stomach, a defence of this kind will carry with it considerable plausibility. In *Waring's* case, a medical witness was questioned upon this point. The deceased is stated to have died from the effects of arsenic in *four hours;* the coats of the stomach were found ulcerated, but no poison could be detected in the organ. The witness admitted on cross-examination, that it was contrary to all experience that ulceration should be occasioned by an irritant poison in less than four hours; but he nevertheless contended that this was the true cause. On such points we can only be guided by observation; and one case of this kind, is sufficient to place the possibility of ulceration being produced by arsenic within a few hours, beyond all question. Dr. Christison mentions a case observed by Mr. Hewson, where many eroded spots existed on the stomach, although the person died from the effects of arsenic in *five* hours. (On Poisons. p. 340.)

Absence of inflammation.—But are the stomach and intestines always found inflamed in cases of poisoning by arsenic? The answer must be decidedly in the negative. At the trial of *McCracken,* at the Derby Autumn assizes, in 1832, for killing his wife with arsenic, the fact of poisoning was clearly established, and a large quantity of arsenic was found in the stomach of the deceased; but there was no appearance of inflammation, either in this organ or the intestines. In a late number of Rust's Magazin I find the two following cases. A Servant-girl had some arsenic administered to her in chocolate. She was seized with nausea and violent pain in the stomach, and died the same evening. On inspection, there was no remarkable vascularity or inflammation of the stomach;—but arsenic was found in the duodenum. A man was taken ill with vomiting and violent pain in the abdomen after partaking of some soup, and he died from symptoms of poisoning. On inspection, the mucous surface of the stomach presented no morbid change, with the exception of slight redness about the cardia. Arsenic was found in the contents of the intestines. In a case quoted by Flandin from Etmuller, a girl swallowed a strong dose of arsenic, and died twelve hours afterwards, without having vomited, or manifested any symptoms. On inspection, arsenic was found in the stomach, but there was no material lesion of the organ. (Op. cit. i. 234.) Even with symptoms of gastric irritation, well-marked appearances may be wanting. (See Dr. Letheby's case, ante, p. 262.) Occasionally the appearances are so slight, that were not the attention of the examiner specially directed to the fact of poisoning, they would be passed over. (See case by Dr. May, ante, p. 260.) These singular cases appear to show, that arsenic does not exert any local action of a chemical nature, like a corrosive, on the stomach; for the action of corrosives takes place on mere contact, without reference to the state of constitution, or the quantity of poison taken. Medical evidence of poisoning from post-mortem appearances is in such cases entirely wanting;—they are not very common, but still they show, that unless great care be taken in forming an opinion, a case of arsenical poisoning may be easily overlooked. They teach this important fact in legal medicine, that the non-existence of striking post-mortem changes in the alimentary canal, is no proof that the party has not died from the effects of arsenic. When the dose is very small, well-marked post-mortem changes are very rarely met with.

In a few rare instances, the mouth, pharynx and œsophagus have been found inflamed, but in general there are no post-mortem changes in this part of the alimentary canal to attract particular attention. The mucous membrane of the *small intestines* may be found inflamed throughout, but commonly the inflammatory redness is confined to the duodenum, especially to that part joining the pylorus. Of the large intestines, the rectum appears to be the most prone to inflammation. The heart, brain and lungs present no appearances which can be considered characteristic of arsenical poisoning. The same remark applies to the liver, spleen and kidneys, although these, like the other soft organs, become recepticles of the absorbed poison.

It is worthy of remark in relation to the known antiseptic properties of arsenic, that the parts specially affected by this poison, (the stomach and intestines,) occasionally present the well-marked characters of irritant poisoning for a long time after death. This was established in the case of the *Queen* v. *Dazley*, tried at the Bedford Summer Assizes, July, 1843. The prisoner was convicted of poisoning her husband with arsenic, upon evidence obtained by the exhumation and examination of the body six months after interment. The stomach and intestines were the only parts of the body undecomposed. This case presents many important subjects for reflection to the medical jurist; as for example, the substitution of arsenic for medicine,—the length of time after death at which good evidence may be obtained from the body,—the fact of another person labouring under symptoms of poisoning by arsenic, who had accidentally partaken of the supposed medicine—and lastly, the evidence from the death of an animal which had swallowed some of the matter vomited by the deceased. In two cases of recent occurrence (*Chesham*) referred to me by Mr. Lewis, coroner for Essex, a deep red inflammatory appearance of the mucous membrane immediately below a layer of sulphuret of arsenic was well-marked, although the bodies had been buried *nineteen* months. This antiseptic power is only likely to be exerted where the solid arsenic remains in the body in large quantities. Absorbed arsenic does not appear to be in sufficient quantity to prevent decomposition.

QUANTITY REQUIRED TO DESTROY LIFE.

This is an important medico-legal question. According to a case quoted by Dr. Christison, the smallest fatal dose on record, in an adult, is stated to have been *thirty* grains of the powdered white arsenic: the man died in six days. But undoubtedly a much smaller quantity than this would kill. Facts of this description can of course only be elicited by accident, as, in cases of suicide or murder, so much more of the poison, than is necessary, is commonly taken. Dr. Christison quotes a case, in which a child four years old, took four grains and a half of arsenic in solution, and died in six hours. (Op. cit. 295.) In *Waring's* case, it was highly probable from the medical evidence that the deceased, an old woman of seventy, was killed by a dose of *four* grains. In a case that fell under my notice, I have reason to think that a young lady was killed by eating a portion of cake which could not have contained more than four grains of arsenic, and probably less than three grains. There is no doubt that very small doses of this poison are capable of producing serious effects; and that some constitutions may be more affected by it than others. It is often safely given in medicinal doses of from 1-16th to 1-8th of a grain; but it is impossible to give half a grain without producing some of the symptoms of poisoning. Dr. Burne has reported the case of a young female, who took only the twentieth of a grain daily for four days, making *one-fifth* of a grain of arsenic. Symptoms of inflammation of the stomach and alarming symptoms of a nervous character appeared, which endangered the patient's life, and rendered a discontinuance of the medicine absolutely necessary. (Med. Gaz. xxv. 414.)

The following case occurred in London, in October 1839. At a large dinner party, it was observed that three persons, who had partaken of port wine on the table, were seized with symptoms of poisoning. The wine was suspected to contain poison, and it was sent to me for examination. It was clear, of the usual colour and odour, and possessed all the characters of good wine; but there was a small quantity of a reddish white sediment at the bottom of the bottle. From the account of the symptoms, the wine was suspected to contain arsenic:—this was found to be the case, and the quantity of poison dissolved amounted to about 1-2 grain in each fluid ounce. The following were the facts. A child about sixteen months, took a quantity of the wine, containing about *one-third of a grain* of arsenic. In twenty minutes this child became sick, vomited violently for three hours, and then recovered. A lady, aged 52, took a quantity of wine, containing rather less than *two grains* of arsenic. In about half an hour, she experienced faintness. Violent vomiting came on, and lasted four hours, but there was no pain. She then gradually recovered. A gentleman, aged 40, took a quantity of the wine, containing rather more than *two grains* of the poison. The symptoms in him were similar, but more severe; and had he taken another glass of the wine, it is probable that he would have been killed. It may be proper to observe, that although this wine was perfectly saturated with arsenic, not the least taste was perceived by any of the parties.

This case shows that two grains of arsenic have been taken without causing death, but it is not thence to be inferred that two grains, or even less, may not suffice to destroy life. As Dr. Christison justly remarks, the two adults may have here owed their escape to the fact, that the poison was taken on a full stomach, and that there was violent vomiting; but this observation cannot apply to the child. From the symptoms produced, we shall certainly be warranted in asserting, that a dose of *three grains* is very likely to prove fatal to an adult. According to Dr. Lachése, from one to two grains may act fatally in a few days:—this, however, is a speculative statement. (Ann. d'Hyg. 1837, i. 334.) It is highly probable that this dose would prove fatal to a child, or to weak and debilitated persons. The opinion respecting the fatal dose, expressed in a former work (Manual of Med. Jurisp. p. 128,) has been strongly corroborated by a case recently reported to the Pathological Society of London, by Dr. Letheby. In this instance, *two grains and a half* of arsenic, contained in two ounces of Fly-water, killed a robust healthy girl, aged nineteen, in thirty-six hours. (Med. Gaz. xxxix. 110.) This I believe to be the smallest fatal dose on record; and it will justify a medical witness in stating that under circumstances favourable to its operation, the fatal dose of this poison is from *two to three grains*.

Recovery from large doses.—Persons have recovered after having taken very large doses of this poison. M. Bertrand states, that he swallowed five grains of arsenious acid with impunity. (Christison, 362.) The poison was here mixed with a large quantity of charcoal. A case is reported, in which sixty grains were taken by a physician, who recovered without suffering very severely. (Med. Gaz. xi. 771.) In another instance, a person recovered after having taken half an ounce of arsenic. The stomach-pump was not used, and the arsenic appears to have been carried off by vomiting and purging. (Med. Gaz. xix. 238.) In Dr. Feital's case, there was *no vomiting*, and yet the individual recovered after having taken half an ounce! (p. 87, ante.) The nature of the treatment in this instance shows that it could not have aided the recovery. A case is quoted by Wibmer, from an American journal, in which a man is reported to have recovered in three or four days after having taken one ounce and a half of arsenic. There was violent vomiting. (Arzneimittle, i. 278.) Cases of recovery when large doses have been taken are not very common. They must be regarded as exceptions to the general rule. It would be in the highest degree improper to infer from them, that a large dose of this poison may be taken with impunity. In these instances, we commonly find either that the arsenic has been taken on a full stomach, or, under appropriate treatment, it has been speedily ejected by vomiting and purging.

PERIOD AT WHICH DEATH TAKES PLACE.

Large doses of arsenic commonly prove fatal in from eighteen hours to three days. Probably, the average time at which death takes place is twenty-four hours. But the poison may destroy life within a much shorter period than this. There are now many well-observed cases reported, in which death has taken place in from three to six hours. I have recently (1845) met with a well-marked case of death from arsenic in five hours. (For another case, see Ann. d'Hyg. 1837, i. 339.) It is singular that a few years since, observations were so limited, that it was thought to be impossible for arsenic to destroy life in a shorter period of time than seven hours! (See ante, p. 96, *Russell's case;*) and this rapidity of death was actually considered as a medical fact, which in some measure tended to negative the allegation of death from arsenic! One of the most rapidly fatal cases on record, I believe to be that which occurred to Mr. Foster, (ante, p. 263.) This gentleman satisfactorily ascertained that the subject, a child under three years of age, died within *two hours* from the effects of arsenic. The quantity taken could not be determined; but the time at which death takes place, is by no means dependent on the quantity of poison taken. Dr. Borland, who formerly attended my lectures, communicated to me the following interesting case, in which death probably occurred in less than two hours. A young married female, of a delicate nervous temperament, bought *two ounces* of arsenic. The whole or greater part of this she swallowed dry, and washed it down with some milk procured on her way home. It would not have occupied more than from fifteen to twenty minutes to go from the place where she purchased the poison to her own house. She immediately stated to her friends that she had taken poison. A medical man residing near was sent for, and attended directly. A stomach-pump was procured, and in the meantime, gum water was given to her with other remedies, and attempts were made to produce vomiting by tickling the fauces. The contents of the stomach were evacuated by the pump, and soap and water injected several times and again withdrawn. She was observed to become pale and faint, and if she had not been prevented, would have fallen from her chair. She was carried to a bed in the room, and instantly expired. The time probably occupied in sending for the stomach pump, and its introduction, might have been about half an hour. Dr. Borland considered that the period from the time of the deceased taking the poison until her death, must have been less than *two hours*. There was no vomiting, except of a little glairy matter, from the artificial means employed, neither had the bowels been acted on from first to last, nor could it be ascertained that she had suffered much pain. The body was not inspected. A case of poisoning by arsenic, but somewhat doubtful, is reported by Metzger to have proved fatal in *half an hour*. The patient died in convulsions. (System der Ger. Arzneim. 256.) In some of these instances of rapid death, the brain and nervous system have been observed to be affected;—the patient suffering from narcotism and convulsions: but this by no means implies that symptoms of irritation are always absent. In Mr. Soden's case (p. 261,) in which not less than four, and probably six, ounces of the poison had been taken, the patient died in less than four hours, and two ounces of arsenic were found in the stomach. We have here an instance, which occurred in March 1810, of arsenic destroying life and producing excessive inflammation in less than *four* hours: and yet at a criminal trial, sixteen years afterwards (Lewes Assizes, 1826,) it was a debated question with some of the medical witnesses, whether it was possible for a person to die from the effects of arsenic in less than *seven* hours, and respectable medical authorities were actually quoted against this view! (p. 96.) Such is the danger of a Court of justice relying for medico-legal facts of this description, upon the personal experiences of witnesses.

An interesting case has been recently published by Dr. Dymock. A girl, aged twenty, took two ounces of powdered arsenic, and died in less than two hours and a half afterwards. There were no comatose symptoms:—the girl was sensible to the last, and she had vomited violently. The mucous membrane of the stomach was covered with bright patches of a scarlet colour. (Ed. Med. and Surg. Journ. April 1843.) In thirteen cases of poisoning by arsenic recorded by Dr. Beck, the smallest quantity taken was one drachm, and the largest two drachms. The shortest period for death was four hours, the longest two days. (Dubl. Med. Press, May 1845.) In some of these rapid cases of death, especially in those of Mr. Foster (Case 2) and that of Dr. Borland, it is evident from the symptoms that the brain and heart had become remotely affected within the very short period of two hours. It is therefore reasonable to infer, that the poison had become absorbed, and that the theory of certain French toxicologists, who assign a period of several hours as absolutely necessary to the action of arsenic, is unfounded. Either the theory is false, or the remote effects of arsenic may be produced independently of absorption! It would be advisable that in these cases of rapid death, the soft organs and tissues should be hereafter examined, in order to determine the earliest period after the administration at which they become penetrated with the poison. In the living person this may be done by the examination of the urine, (ante, p. 30.)

Influence of quantity.—With respect to the effect of quantity, I have known one case prove fatal in fifteen hours where forty grains had been taken; and in another, where an ounce (twelve times the above quantity) had been swallowed, the patient did not die for seventeen hours. Both patients were females of about the same age. It is a common opinion that large doses only, kill with great rapidity; but this is not uniformly observed. In one instance, two ounces of the poison destroyed life in three hours and a half; but in another case (*Waring*) a dose of four or five grains killed a person in four hours. It is obvious that a patient who recovers from the first effects, may still die from exhaustion or other secondary consequences, many days or weeks after having taken the poison. In one criminal case in which I was consulted, the child did not die from the effects of arsenic until after the lapse of two days. In the case *Reg.* v. *M'Cormick*, Liverpool Winter Assizes, the child died, as it appeared, from one dose of arsenic, after the lapse of twelve days. (Med. Gaz. xxxiii. 434.) The child partially recovered from the first effects. In the case of the *Queen* v. *Gilmour* (Edinburgh, Jan. 1844,) the deceased died after thirteen days. In one instance, already mentioned, arsenic was applied externally to the head, and the person did not die until the *twentieth day*, (p. 264.) The longest duration of a case of poisoning by arsenic which I have met with, is reported by Belloc. A woman, aged 56, employed a solution of arsenic in water to cure the itch, which had resisted the usual remedies. The skin became covered with an erysipelatous eruption, and the itch was cured, but she experienced severe suffering. Her health gradually failed, and she died after the lapse of *two years*, having suffered during the whole of this period from a general tremor of the limbs. (Cours de Méd. Lég. 121.)

TREATMENT.

If vomiting does not already exist as a direct effect of the poison, sulphate of zinc should be exhibited, and its emetic effects promoted by mucilaginous drinks, such as linseed-tea, milk, or albuminous liquids. When sulphate of zinc cannot be procured, a good substance for an emetic is powdered mustard, in the proportion of from one to two teaspoonfuls in a glass of water, administered at intervals. A saponaceous liquid, made of equal parts of oil and lime-water, may also be given. While this invests the poison, the lime tends to render less soluble, that portion of the poison which is dissolved. The stomach-pump may be usefully employed; but unless the patient is seen early, remedial means are seldom attended with success. I have known death to occur in a case where every particle of poison was found, on subsequent examination, to have been removed from the stomach. There are many instances of recovery on record, in which the arsenic appears to have been early ejected by constant vomiting and purging. The recovery has, however, been commonly attributed to the supposed antidote. Mr. Tubbs informs me that, conjoined with the use of the stomach-pump and emetics of sulphate of zinc, he has found great service in a mixture of milk, lime-water, and albumen. Such a mixture is undoubtedly well fitted to envelope the particles of arsenic, and sheathe the coats of the stomach from the irritant action of the poison. This gentleman has sent me the reports of no less than nine cases, some of them of a very severe kind, which he has thus successfully treated. For reasons elsewhere stated (ante, p. 78,) I do not think that, in the majority of cases of arsenical poisoning, the slightest confidence can be placed in the hydrated sesquioxide of iron as a *chemical* antidote.

[On the other hand, the experiments of Borelli, Boulay, Damaria, Orfila, Soubeiran, Van Speze and others, show that more reliance is to be placed on this antidote, than is accorded to it by Mr. Taylor. That it is capable of acting chemically is proved by the case given by Devergie (*Med. Leg.* ii. 729.) At the same time it is always of importance to evacuate the contents of the stomach as speedily as possible.—G.]

As a bulky mass it may serve mechanically to suspend the poison, and thus facilitate its ejection from the stomach; but in this respect it possesses no advantages over albumen or other viscid liquids. Our treatment must be directed to the entire expulsion of the poison from the alimentary canal, (p. 73, ante.) Orfila has recommended that diuretics should be employed, in order to promote the secretion of urine, and thus favour the more speedy elimination of the poison from the system. It appears to me that there are some objections to this mode of treatment: it involves the necessity that the whole of the poison should pass into the blood for the purpose of elimination; and further, that it should not be carried off gradually, as would be the case if diuretics were not administered, but that the process of absorption should be expedited by creating a drain upon the kidneys. The danger, however, in the absorption of poisons, appears to arise less from the absolute quantity taken up by the blood, than the quantity admitted into the circulation at any one time. There are various experiments on the absorption of poisons, the results of which confirm this view. (See ante, p. 30; also Flandin, i. 591.) Further, poisons are not always or necessarily eliminated by one secretion. Sometimes they escape more readily by the skin, at others by the saliva. (See IODIDE OF POTASSIUM, ante, p. 230.) It does not therefore follow that the promotion of the urinary secretion would always, *pro tanto*, promote the expulsion of the poison. The researches of M. Flandin show that the quantity of arsenic which escapes by the kidney is exceedingly minute. (See ante, p. 39.) These appear to me to be serious objections to a plan of treatment which implies the saturation of the whole of the blood with poison in as short a time as possible. When arsenic is entirely expelled from the alimentary canal, there can be no injury in the employment of diuretics; but the question would then be, whether the patient would not recover just as readily without them. So long as any poison remains in the stomach or viscera, diuretics are likely to do more harm than good; and when all the poison is expelled, they appear to be useless. The diuretic plan entirely failed in the experiments performed on animals by M. Flandin, i. 568; (also p. 88, ante.)

CHAPTER XXIV.

ARSENIC CONTINUED—CHEMICAL ANALYSIS—TESTS FOR ARSENIOUS ACID IN THE SOLID STATE AND IN SOLUTION—REDUCTION-PROCESS—DELICACY OF THE LIQUID TESTS—SULPHURETTED HYDROGEN—OBJECTIONS TO THE TESTS—SUMMARY OF THEIR VALUE—MARSH'S PROCESS—HYDROGEN TEST—IS ARSENIC A CONSTITUENT OF THE BODY?—REINSCH'S PROCESS—ITS DELICACY—ARSENIC IN LIQUIDS CONTAINING ORGANIC MATTER—DETECTION OF THE POISON IN THE CONTENTS OF THE STOMACH—IN THE TISSUES OF THE BODY—VARIOUS PROCESSES FOR ITS SEPARATION—DISAPPEARANCE OF THE POISON FROM THE BODY—ITS DETECTION IN EXHUMED BODIES AFTER MANY YEARS—ITS ALLEGED EXISTENCE IN THE SOIL OF CEMETERIES—ARSENIC IN SOLIDS—QUANTITATIVE ANALYSIS—ARSENITE OF POTASH—METALLIC ARSENIC—FLY-POWDER—FLY-WATER—ARSENIC ACID—ARSENIATE OF POTASH—SULPHURETS OF ARSENIC—ARSENURETTED HYDROGEN.

CHEMICAL ANALYSIS.

Arsenic as a solid.—In the *simple state*, *white arsenic* may be identified by the following properties:—1. A small quantity of the powder, placed on platina foil, is entirely volatilized at a gentle heat (370°) in a white vapour. Should there be any residue, it is impurity; sometimes plaster of Paris or chalk is found mixed with it. The quantity of fixed impurity present may in this way be easily determined. If a small portion of the white powder be very gently heated in a glass tube of narrow bore, it will be sublimed, and form a ring of minute octohedral crystals, remarkable for their lustre and brilliancy. It will be observed in these experiments, that white arsenic in vapour possesses no odour. 2. On boiling a small quantity of the powder in distilled water, it is not dissolved, but it partly floats in a sort of film, or becomes aggregated in small lumps at the bottom of the vessel. It requires long boiling, in order that it should become dissolved and equally diffused through water, (p. 255, ante.) This was a point of some importance in the case of *Reg.* v. *Lever*, Central Criminal Court, June 1844. A question here arose, whether arsenic would float on tea. I have observed that the film formed on putting powdered arsenic into a vessel of cold water, remained for five weeks on the surface, notwithstanding the occasional agitation of the vessel. On adding a few drops of caustic potash to the mixture of arsenic and water, and applying heat, the poison is entirely dissolved, forming a clear solution of arsenite of potash. 3. When the powder is treated with a solution of hydrosulphuret of ammonia in a watch-glass, there is no change of colour, as there is with most metallic poisons: on heating the mixture, the white powder is dissolved; and on continuing the heat until the ammonia is expelled, a rich yellow or orange-red film is left (sesquisulphuret of arsenic,) which is soluble in all alkalies, and insoluble in muriatic acid.

Reduction-process.—When a small portion of the powder, *i. e.* from one-fourth to one-twentieth part of a grain is heated with some reducing agent containing carbon, in a glass tube about three inches long and one-eighth of an inch in diameter, it is decomposed: a ring of metallic arsenic of an iron-grey colour is sublimed and deposited in a cool part of the tube. At the same time there is a perceptible odour, resembling that of garlick, which is possessed by metallic arsenic only while passing from a state of vapour to arsenious acid. This *odour* was at one time looked upon as peculiar to arsenic, but no reliance is now placed on it as a matter of medical evidence—it is a mere accessory result. Many mistakes were formerly made respecting it. Thus, we find it stated to have been perceived under circumstances in which it could not possibly have been produced! (Marshall on Arsenic, 90, ed. 1817.) It was not then known that white arsenic (arsenious acid) possessed no odour in the state of vapour. In this experiment of reduction, there are commonly two rings deposited in the tube, the upper of which has a brown colour, and appears to be a mixture of finely divided metallic arsenic and arsenious acid. It has been regarded by some chemists as a sub-oxide, more volatile than the metal. Various reducing agents have been proposed: for example, charcoal, black flux, calcined cream of tartar, the oxalate of lime or soda, the formate of soda; but that which I have found most convenient, is the residue of the tartrate or acetate of soda (incinerated in a covered platina crucible,) which consists of carbon and carbonate of soda. It does not deliquesce, and may be kept for years without change, (ante, p. 122.) The proportion in which it should be employed is about two or three parts of flux to one of white arsenic. Cyanide of potassium has been lately recommended; it answers very well, but it is apt to become moist. I have found that gallic acid is also a good deoxidizing agent, but it is not equal to the soda-flux. In order to determine the *weight of the sublimate,* the glass tube should be filed off closely on each side of the metallic ring, and weighed; the sublimate may then be driven off by heat, and the piece of glass again weighed:—the difference or loss represents the weight of the sublimate. These sublimates are remarkably light, and require to be weighed in a delicate balance. I found, in one experiment, a large sublimate to weigh no more than ·08 grains. By heating gently the piece of tube, reduced to powder in an agate-mortar, in another tube of larger diameter, the metallic arsenic, during volatilization, forms octohedral crystals of arsenious acid, which may be dissolved in a few drops of water, and tested. When the quantity of arsenious acid is so minute as to be scarcely ponderable, it would be advisable to employ for its reduction finely powdered and dry charcoal, since the alkali in the soda-flux might retain the whole or the greater part of the arsenic in combination. The minute quantity of arsenious acid should be dropped into a dry and warm tube, not more than the eighth or the tenth of an inch in diameter, and the charcoal, well dried, dropped upon it in the proportion of three or four times its bulk. The upper part of the charcoal should be brought to a high temperature before the arsenic is heated. In this way, distinct arsenical sublimates may be procured, weighing considerably less than the 1000th part of a grain. The delicacy of this test cannot, however, be estimated by the weight of the sublimate, but by the weight of the arsenious acid on which we can operate. Dr. Christison states, that a distinct metallic sublimate may be obtained from the 300th part of a grain, (op. cit. 260.) These sublimates may be preserved unchanged for years by filing off the ends of the tube, and then hermetically sealing them in the flame of a spirit-lamp.

Objections to the reduction-process.—Corrosive sublimate is volatile like white arsenic, but it differs from it in all its other properties. It is very soluble in water, insoluble in potash, which turns it of a yellow colour,—while hydrosulphuret of ammonia turns it black. Indeed, it may be said that there is no substance but arsenic which possesses the *three* first characters mentioned; they should, however, be taken together. With regard to the fourth character, namely, the production of a metallic sublimate, numerous objections have been made:—1. The glass itself may acquire a black metallic lustre by heat from the reduction of the *oxide of lead* contained in it. This is always the case when the tube is held too much in the body of the spirit-lamp flame instead of over the point. This metallic stain differs in appearance from arsenic; it is fixed while the arsenical sublimate is volatile by heat, and convertible to white octohedral crystals of arsenious acid. 2. *Charcoal* may give a dark colour to the tube, but it is not advisable to employ this substance unless the quantity of arsenious acid be very minute; besides, the stain of charcoal is fixed, and has no metallic lustre like that of arsenic. 3. Arsenic is said to be contained in *glass*, and it was supposed that it might be sublimed by heat; this, however, is impossible: arsenic is sometimes used in the manufacture of glass, but it is entirely volatilized during the process. (See Ann d'Hyg. 1834, i. 224; also Galtier, Toxicologie, i. 297.) I have frequently examined large quantities of glass-tubing employed by chemists in a finely powdered state, without finding the slightest trace of arsenic. 4. *Cadmium* is a metal which is said to form a metallic sublimate like arsenic. The oxide of cadmium may be reduced by a similar process, but the metallic sublimate is wholly different from that of arsenic: it has a tin-like lustre, and is generally fringed with a brown margin of reproduced oxide. There is no odour of garlic during the reduction of oxide of cadmium; and on heating the metallic ring, it is not wholly volatilized like arsenic, but converted to a ring of brown oxide. Oxide of cadmium is of a brown colour, it cannot be volatilized on platina by the heat of a spirit-lamp, it is quite insoluble in potash, but easily dissolved by nitric acid. If there were no perceptible difference in the sublimates produced by the two bodies, these characters would at once form a clear distinction between them. Oxide of cadmium is moreover a rare substance; it is difficult to meet with it. 5. Mercury forms a sublimate, but in white silvery globules, quite distinct from the dark iron-grey lustre of arsenic. Neither antimony nor zinc can be volatilized from any of their preparations in a metallic state, by the heat of a spirit-lamp. *The process of reduction*, with the most simple precautions, is, therefore, when thus applied, conclusive of the nature of the substance under examination. It is advisable, although not absolutely necessary, that we should apply the three foregoing tests to the white powder, before attempting to extract the metal from it.

With respect to the other properties of arsenious acid, it may be remarked, —that it is very soluble in boiling muriatic acid, if concentrated; and by this means it may be partially separated from the sesqui-sulphuret or orpiment, which is not dissolved by that acid. The solubility of arsenious acid in muriatic acid, aids the deposition of the metal on copper in REINSCH'S PROCESS (p. 352, post.) It is not dissolved by nitric acid, but is oxidized by it on long boiling, and converted to arsenic acid; and lastly, it is soluble in alcohol, and is not precipitated by this reagent from liquids in which it is dissolved. The presence of neutral salts does not appear to affect materially its solubility in water; but nitre is said to render it more soluble, (p. 255, ante.)

Arsenic in solution in water: Liquid tests.—The aqueous solution of arsenic is clear, colourless, possesses scarcely any perceptible taste, and has a very faint acid reaction. In this state, we should first evaporate slowly a few drops on a glass plate, when a confused crystalline crust will be obtained. On examining this crust with a common lens, it will be found to consist of numerous minute octohedral crystals, presenting triangular surfaces by reflected light. By this simple experiment, arsenic is distinguished from every other metallic poison. 1. On adding to the solution,—*Ammonio-nitrate of silver,*—a rich yellow precipitate of arsenite of silver falls down:—rapidly changing in colour to a greenish brown. The test is made by adding to a very strong solution of nitrate of silver, a weak solution of ammonia, continuing to add the latter, until the brown oxide of silver, at first thrown down is almost re-dissolved. The yellow precipitate is soluble in nitric, tartaric, citric, and acetic acids, as well as in caustic ammonia. It is not dissolved by potash or soda. 2. On adding to the solution of arsenic, *Ammonio-sulphate of copper,* a rich green precipitate is formed, the tint of which varies, according to the proportion of arsenic present and the quantity of the test added; hence, if the quantity of arsenic be small, no green precipitate at first appears: the liquid simply acquiring a blue colour from the test. In less than an hour, if arsenic be present, a bright green deposit is formed, which may be easily separated from the blue liquid by filtration. This test is made by adding ammonia to a weak solution of sulphate of copper, until the blueish-white precipitate, at first produced, is nearly redissolved; it should not be used in large quantity if concentrated, as it possesses a deep violet blue colour, which renders obscure the green precipitate formed. The precipitated arsenite of copper is soluble in all acids, mineral and vegetable, and in ammonia, but not in potash or soda. When dried and collected, it possesses this characteristic property:—by very slowly heating a few grains in a tube of small bore, arsenious acid is sublimed in a ring of minute resplendent octohedral crystals,—oxide of copper being left as a residue.

Objections to the liquid tests.—These are called the liquid tests for arsenic. The *Silver* test, first discovered by Mr. Hume, in 1789 (Marshall on Arsenic, 87,) acts with remarkable delicacy, and is of great use as a corroborative test in the various processes for determining the presence of this poison. A solution of an *alkaline phosphate,* which yields a yellow precipitate with nitrate of silver, is not affected by the ammonio-nitrate when properly made; and conversely, a solution of arsenious acid gives only a faint turbidness with nitrate of silver, while it is copiously precipitated of a yellow colour by the ammonio-nitrate. Medical jurists appear to have overlooked the fact, that a diluted solution of *phosphoric acid* may be, in some cases, precipitated by this test, exactly like a solution of arsenic; but the answer to any objection on this ground, is that pure phosphoric acid either gives no precipitate, or one of a pale blue colour, with the ammonio-sulphate of copper,—that it is not affected by sulphuretted hydrogen gas, and lastly, that on boiling copper in the acid liquid, and adding muriatic acid, there is no deposit of arsenic on this metal. Phosphorus, it must be remembered, may contain arsenic, and thus contaminate the preparations into which it enters. (See case, Med. Gaz. xxxv. 655.) With respect to the delicate reaction of this most useful test, Mr. Marshall states (On Arsenic. p. 94, ed. 1817,) that it is fully capable of detecting the 1000th part of a grain in solution,—a proof that the application of this test was well understood more than a quarter of a century ago. Dr. Traill has lately asserted that the 16,000th part of a grain of arsenic in solution, is precipitated by the silver-test, and that with the 10,000th part of a grain the precipitate is visible to the eye. I have found that the 8,000th part of a grain dissolved in one drop of water, gave a pale yellow film; but the result materially depended on the quantity of water present. Thus the 4,000th part of a grain of arsenic in ten drops of water, was not perceptibly affected by the test; but the 2,000th of a grain dissolved in four drops of water, gave a decidedly yellow precipitate. The evidence derivable from these minute reactions would not be of much value, except that the test is used to corroborate inferences from the results of other experiments. The sulphate of copper test is far less delicate in its reaction, and having an intensely blue colour, it entirely conceals the green tint which may be given by a small quantity of precipitated arsenite of copper. Thus it was found in an experiment, by cautiously adding an arsenical solution of known strength to a few drops of the test, that no green tint appeared in the precipitate, until the quantity of arsenious acid amounted to the 173rd part of a grain in less than one fluid-drachm of water —the degree of dilution being about 8,640 times. Whenever the arsenic is in small quantity in its aqueous solution, not more than one or two drops of the ammonio-sulphate should be added by means of a glass rod.

No one, in the present day, would think of employing these liquid tests in solutions, in which the arsenic was mixed with *organic matter*. Almost all liquids used as articles of food, are precipitated or coloured by one or both of them, somewhat like a solution of arsenic, although none of this poison be present. Thus, then, any evidence founded on their employment, unless the arsenic be dissolved in pure water, or unless the precipitates be proved to contain the poison, should be rejected. These liquid tests are now employed rather as adjuncts to other processes, than as a direct means of detecting arsenic. An exclusive reliance upon them has led to the rejection of chemical evidence on several trials, where they had been most improperly employed in the analysis of suspected liquids containing organic matter. The trial of *Donnall* at Launceston, in 1817, affords a memorable lesson to the medical jurist on this subject. (Smith's Anal. of Med. Ev. p. 212.)

It would be unsafe for the analyst to trust merely to the production of a green precipitate by the addition of the *Copper test*, as a proof of the presence of arsenic. Several colourless organic acids, as the acetic and the malic, give a green colour with the test; and some mixtures of substances may give with it a precipitate so closely resembling arsenite of copper, as to render a distinction by the mere appearance, impossible. Mr. Tubbs, to whose researches on arsenic I have elsewhere alluded, forwarded to me a liquid which had been procured by carbonizing with sulphuric acid, blood taken from a person labouring under the effects of arsenic, and digesting the ash in nitric acid. The liquid was acid, owing to the presence of nitric and sulphuric acids. It gave a grass-green precipitate with the ammonio-sulphate of copper, precisely resembling Scheele's green; but there was no arsenic present, since neither the silver nor the sulphuretted hydrogen test gave the characteristic results; nor did the precipitate contain arsenic. The deceptive effect was due to the complex nature of the liquid. Iron (from the blood) was evidently dissolved in it, and phosphates (derived from the blood) were also present. In an artificial mixture of this kind, without iron, a blue precipitate is formed by the test; but if a persalt of iron be present, the precipitate is green. Again, a persalt of *Uranium* gives with the *Copper test* a grass-green precipitate, closely resembling that produced in a solution of arsenious acid. With the ammonio-nitrate of silver the uranium salt gives a yellowish precipitate, passing speedily to a dingy brown colour; but the uranium salt, among other properties, is easily known by a solution of ammonia giving with it a yellow precipitate of oxide of uranium.

The correction of fallacies of this kind depends—1. On not trusting to the action of *one* test only. 2. On not trusting to *colour* but applying tests to the precipitate. If the green precipitate be *arsenite* of copper, it should yield octohedral crystals when gently heated, or give a grey deposit of metallic arsenic on copper when boiled with muriatic acid slightly diluted. (See **Reinsch's process**, p. 286.)

3. *Sulphuretted hydrogen gas.*—The hydro-sulphuret of ammonia gives no precipitate in a solution of arsenic until an acid has been added, whereby arsenic is known from most metallic poisons, On adding an acid (acetic) a rich golden yellow-coloured precipitate is thrown down (orpiment or sesqui-sulphuret of arsenic.) It is better, however, to employ in medico-legal analysis, a current of washed sulphuretted hydrogen gas, which is easily procured by adding sulphuret of iron to one part of strong sulphuric acid and three parts of *water* in a long-necked bottle, (see ante, p. 122.) The arsenical liquid should be slightly acidulated with acetic or very diluted muriatic acid, before the gas is passed into it: at least care should be taken that it is not alkaline. The yellow compound is immediately produced if arsenic be present, and may be collected after boiling the liquid so as to drive off any surplus gas. The precipitation is likewise facilitated by adding to the liquid a solution of muriate of ammonia. This yellow precipitate is known to be sesquisulphuret of arsenic by the following properties:—1. It is insoluble in water, alcohol, and ether, as well as in all acids mineral (muriatic) and vegetable; but it is decomposed by strong nitric and nitro-muriatic acids. 2. It is immediately dissolved by caustic potash, soda, or ammonia, forming, if no organic matter be present, a colourless solution. 3. When dried and heated with three parts of soda-flux, or, what is better, an equal part of cyanide of potassium, it furnishes a metallic sublimate of arsenic. This last experiment requires a little care, as some sulphur is apt to be sublimed, and obscure the results. If fine pulverulent silver be used as the reducing agent, and the heat *gently* applied, the arsenic is evolved at once from the sulphuret in a ring of octohedral crystals of arsenious acid. Unless these properties are proved to exist in the yellow precipitate formed by sulphuretted hydrogen in an unknown liquid, it cannot be a compound of arsenic; and it would not be safe to receive evidence on the point. On the other hand, when these properties are possessed by the precipitate, it must be arsenic, and can be no other substance. This test is extremely delicate in its reaction. It begins to give a yellow tinge when the liquid contains only the 4,000th part of a grain of arsenious acid in ten drops of water; the arsenic therefore forming about the 40,000th part of the solution. This becomes more decided with the 2,000th part of a grain, and still more with the 250th part of a grain: the sesquisulphuret is not, however, precipitated until a solution of muriate of ammonia, in which it is insoluble, has been added to the liquid. It is important to observe that the effect produced by the test will materially depend on the quantity of water in which the given weight of arsenic happens to be diffused. In one experiment the gas was passed into a solution containing the 400th part of a grain in twenty drops of water: the results were clear and decided; the liquid acquired a rich golden, yellow colour, but when passed into a solution containing the same weight of arsenic in half an ounce of water, a yellow tint was scarcely perceptible. The arsenic in the first case was in the proportion of the 8,000th, and in the second of only the 1,000,000th part of the solution.

[According to Devergie the relative delicacy of these tests is—ammonio-sulphate of copper, 5,200—sulphuretted hydrogen 200,000—ammonio-nitrate of silver 400,000.—G.]

Objections to the gaseous test.—Many objections have been taken on criminal trials to the medical evidence, founded on the application of this most valuable test. 1. *Cadmium.* It is remarkable that this metal should furnish, at the same time, a plausible ground of objection, both to the process by reduction from the solid state, and to the gaseous test applied to a solution of the poison. Thus the soluble salts of cadmium yield, with sulphuretted hydrogen, a rich yellow precipitate resembling closely that produced by arsenic, and this also gives a metallic sublimate when heated with soda-flux. There are, however, these striking differences;—the yellow compound of arsenic is soluble in ammonia, that of cadmium is insoluble,—the compound of arsenic is insoluble in strong muriatic acid, that of cadmium is perfectly soluble. Of the dried precipitates, the sulphuret of arsenic is not perceptibly affected by stong muriatic acid,—that of cadmium is dissolved readily in the cold with the evolution of sulphuretted hydrogen gas; and a colourless salt of cadmium (chloride) is thereby formed, precipitable as a white carbonate by alkaline carbonates. On *boiling* the sulphuret of arsenic in strong muriatic acid, a very minute portion of sulphuretted hydrogen is evolved, showing that a slight decomposition takes place; but with the sulphuret of cadmium there is immediate decomposition. A solution of a salt of cadmium is immediately thrown down, of a rich yellow colour, by hydro-sulphuret of ammonia,—that of arsenic is not precipitated by this agent. There are many other differences: thus cadmium is not precipitated on copper like arsenic, when boiled with muriatic acid, and it does not combine with hydrogen to form a combustible gas. (See table, ante, p. 130.) An objection on the ground of the strong similarity of cadmium to arsenic, was unsuccessfully taken to the chemical evidence given on the trial of *Mrs. Burdock* at Bristol, in 1835. 2. *Tin.* A persalt of tin is precipitated of a dusky yellow colour by the gas; but the precipitate is destitute of all the properties of sulphuret of arsenic; it is insoluble in ammonia, and it gives no metallic sublimate when heated with flux. A solution of tin is also known from one of arsenic, by its being instantly precipitated by the hydrosulphuret of ammonia. 3. *Antimony.* A solution of this metal is precipitated of a rich orange-red (not yellow) colour by the gas,—the precipitate yields no metallic sublimate with flux, and the solution of antimony is also precipitated by hydrosulphuret of ammonia. 4. *Uranium.* A solution of a persalt of uranium gives, with a current of sulphuretted hydrogen gas, a yellow brown precipitate, wholly unlike that caused by arsenic. This precipitate differs from that of sulphuret of arsenic, in being insoluble in ammonia, soluble in muriatic acid, and in yielding no metallic sublimate with soda flux. Besides, a solution of a uranium salt is precipitated by hydrosulphuret of ammonia.

Although a persalt of uranium gives precipitates with the ammonio-nitrate of silver and the ammonio-sulphate of copper, which in colour somewhat resemble those produced by arsenic, the special characters of uranium are well-marked, and wholly different from those of arsenic. Thus the solution is precipitated yellow by ammonia alone, and of a rich copper red colour by ferrocyanide of potassium. (p. 130, ante.)

It is customary for toxicologists to lay down the rule, that the objection urged against one test for arsenic, is removed by the application of the other tests. In a criminal case in which I had to give evidence (*Reg.* v. *Jennings*, Berks Lent Ass. 1845,) it was ingeniously urged in the defence, that there might perchance be such a mixture of substances not containing arsenic, as to affect all the tests like arsenic when separately applied. This, however, is clearly a chemical impossibility, for it would require the mixture of incompatible substances, such as an alkaline phosphate, a salt of uranium, and a salt of cadmium. But a mere change of colour, or even the production of a coloured precipitate on adding a test to an unknown liquid, furnishes no evidence, *unless the properties of the precipitate be those of an arsenical compound.* Again, no conceivable mixture of substances would produce a metallic ring resembling that of arsenic, so as to deceive one experienced in such matters; and far less a ring, possessing those properties of an arsenical sublimate, which it would be easy for one who may have had but little experience, to determine by simple chemical processes.

Is the reduction-process indispensable to chemical evidence?—An important medico-legal question has arisen in relation to the tests for arsenic, namely,—whether *we can rely upon any tests for this poison, independently of its reduction to the metallic state.* Is it absolutely necessary, chemically speaking, to obtain the *metal* in order to say that arsenic is certainly present in an unknown case? There is a popular prejudice in favour of this metallic reduction; and Courts of law, as well as the public, are disposed to regard the obtaining of the metal, as the only conclusive proof of the presence of this poison. The acquittal of *Donnell*, at Launceston, in 1817, mainly took place from the circumstance that the medical witnesses could obtain no metallic arsenic:—they trusted to the liquid reagents alone, and these had unfortunately been applied to coloured fluids mixed with organic matter. At a trial on the Norfolk Spring Circuit, 1833, the medical witness admitted that the metallic reduction would have been more satisfactory,—but he had consumed the fluids of the stomach in applying the liquid reagents! This evidence, although not absolutely rejected by the Court, was not well received, and the prisoner was acquitted. As this is a purely chemical question, it must of course be answered on chemical principles; for it is chemical certainty that the law requires. If a white powder be presented for analysis, and it is found to possess distinctly the three first characters described (p. 273,) could any chemist entertain a reasonable doubt that the powder was white arsenic?—I think not. The reduction-process might corroborate, but it could not add *greater* certainty to the results thereby obtained; and in heating such a powder with flux, the chemist knows that a metallic sublimate must, of necessity, be formed; for there is no white solid in the whole range of substances known to chemistry, if we except arsenious acid, which possesses the three characters mentioned. If we are so situated that we are obliged to rely upon *one* test only, then the process by reduction should be preferred; but even here, so many mistakes have been made relative to the supposed metallic crust obtained from an unknown solid, that Dr. Turner and others have recommended that it should always be reconverted to arsenious acid in oxidizing it by heat; and that the white solid thus produced, should be tested by liquid reagents. If arsenic in the form of a sublimate were presented to a chemist, and he were required to state its nature, he would necessarily treat it in this way, before expressing a judicial opinion;—because its real nature could only be established with certainty by such experiments. In a case in which the particulars are entirely unknown, there is nothing in the physical characters of an arsenical sublimate, to justify a witness in giving a positive opinion respecting it, before he has submitted it to various chemical processes.

It appears to me that the action of sulphuretted hydrogen and the characters of the resulting sulphuret, coupled with the negative effect of hydrosulphuret of ammonia, as clearly indicate the presence of arsenic, chemically speaking, as the production of a metallic sublimate. When the result is at all doubtful, the sulphuret should be reduced; but, in such a case, if a sublimate be obtained by the reduction of the sulphuret, the precise nature of this should be verified by gently heating it in a wide reduction tube under a free access of air. In a case otherwise doubtful, we should always avoid relying upon *one* test only:—the reduction-process is open to as great fallacies as the gaseous test; and if it be true that there is no other substance in chemistry which yields by reduction a metallic ring like that of arsenic, it is equally true that there is no substance in chemistry which yields with sulphuretted hydrogen gas a golden-yellow precipitate, soluble in potash, soda, and ammonia, and insoluble in muriatic acid. If a person be poisoned by potash, and the alkali is found in the stomach, the medical witness can safely depose to that fact without extracting the potassium; although there is no test applied to potash, which would be so unexceptionable, as the properties possessed by the metal potassium, if it could be readily extracted. But can it be said that chloride of platina and tartaric acid more strongly indicate the presence of potash, than sulphuretted hydrogen and the liquid tests indicate the presence of arsenic? So, in the case of barytes, it is not required as a point of chemical evidence that the metal barium should be reproduced; but—are the liquid tests for barytes more conclusive than the gaseous and the liquid tests for arsenic? If we refer to the common metals themselves, we do not find that this doctrine of metallic reduction is invariably carried out; or the processes of toxicology would, with the exception of a very few cases, consist in simply incinerating the suspected substance with flux and charcoal. On the contrary, this latter process is only resorted to in extreme or doubtful instances; and the sufficiency of the liquid reagents for detecting metals, is made evident by the care bestowed on the description of them by all toxicological writers. In a case of poisoning by lead, tin, zinc, silver, or iron, the presence of each may be speedily demonstrated by the application of liquid tests; and yet these are not more free from fallacy than those for arsenic. Admitting that practical toxicology was confined to the extracting of the pure metals alone, how could a prudent chemist be satisfied of the nature of certain minute discoloured particles of metal without dissolving them in an acid, and trying the solutions with liquid reagents? How could he distinguish a fragment of copper from a fragment of titanium, or tin from cadmium? It appears to me, then, that we may have a chemical certainty of the presence of arsenic, without the production of the metal; and this rule is either rightly applicable to arsenic, or it is most wrongly applied to all other medico-legal cases. It has been stated that the reduction-process is not more conclusive in the opinion of a chemist, than the method by fluid tests; but the former is considered necessary, rather as a concession to the unscientific minds of a criminal court and jury. I fully agree that this principle should always be kept in mind by every medical witness; but I cannot approve the doctrine, that any criminal court should be permitted to select its own degree of *chemical* proof in reference to a subject with which it must be entirely unacquainted. A scientific medical witness ought to be most freely trusted in this, as he is in other more abstruse parts of medical jurisprudence. It must be admitted, however, that since the introduction by Reinsch of the very simple method of reduction by copper, this question formerly so much debated has lost its interest. There is now no reason why a witness should neglect to procure arsenic in the metallic state; since the metal may be easily obtained from a quantity of this substance so small as scarcely to be affected by the liquid tests. The old process is completely reversed—instead of procuring the arsenious acid, and converting it to the metallic state, the metal is now first procured as a deposit and afterwards converted to arsenious acid. (See p. 286.)

MARSH'S PROCESS. HYDROGEN TEST,

The action of this test depends on the decomposition of arsenious acid and its soluble compounds, by hydrogen evolved in the nascent state from the action of diluted sulphuric acid on zinc. The apparatus is of the most simple kind, and is so well known as to need no description. The arsenic may be introduced into the short leg of the tube in the state of powder; but it is far better to dissolve it in water, by boiling, either with or without the addition of a few drops of caustic potash. The metallic arsenic combines with the hydrogen, forming arsenuretted hydrogen gas, which possesses the following properties. 1. It burns with a blueish-white flame, and thick white smoke (arsenious acid.) 2. A cold plate of glass or white porcelain held in the flame near the point, receives a dark stain from the deposit of arsenic upon it. This stain is composed in the centre of pure metallic arsenic, which may be sometimes raised up in a distinctly bright leaf of metal,—immediately on the outside of this, is an opake black ring, (suboxide or hydruret of arsenic,) which, when viewed by transmitted light, is of a clear hair-brown colour at the extreme edge:—if the quantity of arsenic be very small, the metallic lustre and opacity may be wanting, and the whole stain will have a brown colour by transmitted light. On the outside of this black ring, is a thin wide film of a milk-white appearance, which is nothing more than arsenious acid reproduced by combustion. 3. A white saucer or a slip of card or paper moistened with ammonio-nitrate of silver, held about an inch above the point of the flame, will be found, if arsenic be present, to be coloured yellow, from the reproduced arsenious acid being absorbed, and forming yellow arsenite of silver, easily soluble in acetic acid and ammonia. Unless the gas possess these properties, there is no certain evidence of the presence of arsenic in the liquid examined. This process is probably the most delicate of all those which have been devised for the detection of arsenic; but for this very reason, it requires the greatest care in its application. Its delicacy has been sometimes improperly estimated by the assumed weight of the metallic deposit on glass; whereas it is probable that the quantity of arsenic in one infinitesimal deposit, if transferred to the apparatus, would give no indication whatever of the presence of the poison. In this process it must be remembered that in operating on the poison, we are dividing and sub-dividing the metal into a series of deposits, the weight of some of which might not be equal to the millionth part of the weight of the arsenic which is actually furnishing them. More or less arsenic is always lost during the combustion of the gas. M. Villain has lately attempted to determine how many metallic deposits can be obtained from a given weight of arsenious acid. The result at which he arrived is, that 1-65th part of a grain will yield on an average 226 metallic deposits of an average diameter of the 1-12th of an inch. (Journal de Chimie, 1846, p. 611.) The average weight of each, therefore, even supposing there were no loss, would be about the 1-15000th part of a grain.

Objections to Marsh's process.—Other substances will combine with nascent hydrogen, and when that gas is burnt, a deposit will be formed on glass which may be mistaken for arsenic. Late researches have shown, that a liquid containing antimony, tellurium, selenium, iodine, bromine, phosphorus, sulphur, or some kinds of organic matter, may combine with hydrogen, to produce an inflammable gas, and leave a visible deposit or stain on glass. The only objection of any practical force is that founded on the presence of *antimony.* There are these differences between the arsenical and antimonial stains; the stain of antimony has rarely the bright metallic lustre which that of arsenic commonly presents; by transmitted light it is of a smoky black, while that of arsenic is of a hair-brown colour. Although the antimonial is very similar in colour to the arsenical flame, yet the third property is entirely wanting. If the ammonio-nitrate of silver be held over the antimonial flame, the silver is reduced; no yellow arsenite is formed, as in the case of arsenic. This last criterion distinguishes the arsenical flame from that produced by all the other bodies above mentioned.

Deposits of arsenic and antimony.—When the quantity of the liquid yielding the metallic deposit is small, and there is some doubt concerning its real nature, it will be advisable to receive the deposit in a watch-glass or in a thin porcelain capsule. We then add to it two drops of muriatic and one drop of nitric acid,—the metallic film disappears and a brownish-white residue is left on evaporation. If the deposit be entirely *arsenical*, the residue (arsenic acid) is soluble in water, and on adding to it a drop of the nitrate of silver, there will be produced a dingy red precipitate of arseniate of silver. Should it be *antimonial*, the residue (peroxide of antimony) is insoluble in water, and produces no red coloured precipitate on the addition of nitrate of silver. It has been recommended to expose the metallic deposits to the vapours of iodine and phosporus, which affect somewhat differently those of antimony and arsenic: but, from my experiments, the above-described plan is more speedy and satisfactory. The vapour of phosphorus rapidly causes the disappearance of the arsenical, but not of the antimonial deposit. (See APPENDIX.)

It may so happen that we have to deal with a *mixed deposit* of arsenic and antimony: and the great object of the analyst will then be to discover *arsenic* in *antimony*, not antimony in arsenic—as this latter can very rarely become a medico-legal question. The same process should be pursued:—the compound deposit may be dissolved in nitro-muriatic acid, and evaporated to dryness. Arsenic acid may then be dissolved out of the residue by distilled water, and tested by nitrate of silver. If no brick-red precipitate be produced, the deposit could not have contained arsenic. A small white porcelain capsule is serviceable for this experiment, as it permits any change of colour, on the addition of the test, to become immediately preceptible.

Arsenic in Zinc and Sulphuric acid.—The metallic deposit may be proved to be arsenical, and yet it may be fairly alleged that the arsenic was derived from other sources, and not from the suspected liquid. Zinc and sulphuric acid, which are employed in the experiment, are often very impure. Dr. Clark, of Aberdeen, informs me that he has not discovered a specimen of zinc free from arsenic, when about an ounce of the metal was used in an experiment, and the hydrogen gas evolved, was tested by a solution of nitrate of silver. Sulphuric acid has been found to contain either arsenic or selenium; the latter substance yields a brown deposit; but in every other respect it differs from arsenic. The sulphuric acid of commerce sometimes contains a large quantity of arsenic. Mr. Scanlan found that 2000 grains of one specimen of acid yielded 1·5 grains of sesquisulphuret of arsenic. (Pharm. Jour. Aug. 1844.) I lately met with a specimen so impregnated with arsenic as to render it dangerous for use in the preparation of hydrogen. Arsenic may be easily detected in the impure acid by Reinsch's process. The best answer to all objections of this kind is, that the materials should be tried repeatedly, before the suspected liquid is introduced into the apparatus. If no sublimate be formed until after the introduction of the suspected liquid, it is evident that the arsenic must be in the liquid introduced; a fact which may be considered as clearly established, if, on removing the liquid and washing out the tube, no stains whatever result from employing portions of the same sulphuric acid and zinc. If infinitesimal traces be really present in the materials employed, they are obviously, under these circumstances, not to be detected by Marsh's apparatus and can present no practical objection to its use, unless we adopt the most improbable notion that the impurities are brought out in the materials through the action of the test by a mere coincidence at the time of the addition of the suspected poisonous liquids. In using this test, fresh zinc should be employed for each experiment; and the apparatus should be thoroughly cleansed before use. Dr. Geoghegan has found that arsenic is liable to be deposited on zinc by standing; and the same deposit is apt to take place in the fine tube connected with the stop-cock. These are, I believe, the only tangible objections to the use of Marsh's test, and they are not difficult of removal, when moderate care is taken. It will be apparent, that not one of these objections could apply, except to those cases where Marsh's test is relied on, as the *sole* and exclusive chemical proof of the presence of arsenic; but in most instances where this test is *safely* applicable, other tests are also applicable; and it does not at all diminish the merit of this most useful and ingenious discovery, to say that the results which it furnishes, should be corroborated by the use of some of the other tests, if it were only for the sake of preventing any plausible objections to the inference derivable from its use. The great object of chemical evidence is not to show a Court of law what may be done by the use of *one* test only, but to render the proof of the presence of poison most clear and convincing. If, in any case, we have no other evidence to offer, but that furnished by Marsh's test —a case in which the quantity of poison must be infinitesimal, and the metallic deposits proportionably minute,—then it would be better to abandon the evidence altogether, than to maintain that poison is present from results which admit of no sort of corroboration; for all who have experimented on the subject, must have perceived the utter inefficacy of applying liquid tests to determine the chemical properties of imponderable and scarcely visible sublimates. This appears to me to have been the most objectionable part of the evidence in the well-known case of *Madame Laffarge* (1840.) Orfila admitted that he had obtained only a few deposits so slight that they could not be weighed. He estimated the united weight at half a milligramme, or about one-thirteenth of a grain.

Normal arsenic.—Is arsenic a constituent of the body? It was owing to too great confidence in the extreme application of Marsh's process, that arsenic was pronounced to be a natural constituent of the human body, existing especially in the bones, and also probably in the muscular system; owing to the same cause, it was said to exist constantly in the loose soil of cemeteries, and rules were given to distinguish *norml arsenic* from that taken as a poison! The experiments of many English chemists, as well as those performed before the Academy in France by M. Orfila himself, have shown that arsenic does not naturally exist in any part of the body; and that there must have been a fallacy in his previous experiments. The following is an extract from the report published by Orfila:—"*Expériences pour rechercher l'arsenic dans le corps de l'homme à l'état normal.*" Dans douze expériences faites par les commissaires avec la chair musculaire, avec des os plus ou moins calcinés et traités tantôt par l'acide chlorhydrique à l'air libre ou en vaisseaux clos, et avec du bouillon de bœuf, on n'a jamais obtenu d'arsénic (arsénic normal.) (Rapport de l'Académie Royale de Médecine, &c. par M. Orfila, 1841, p. 45.) M. Flandin showed that the effect mistaken for that of arsenic, probably arose from the presence of phosphite and sulphite of ammonia mixed with animal matter (i. 617.) For a full statement of the facts, see Flandin, i. 728. If arsenic were really present in bone, the process of incineration would probably remove it. It is to the employment of this process that M. Legrip ascribes the failure of analysts to detect arsenic in bone. He states that he has found it by dissolving bone in diluted muriatic acid. (Journal de Chimie, Mai 1847, p. 261.) This objection may be, to a certain extent, valid; but it was *after* incineration that Orfila alleged he had discovered arsenic in bone. As the bones are never analysed in practice, it is quite unnecessary to enter into a further discussion of the question.

In 1840, a man was condemned to death in France on a charge of murder by poison *ten years* after the perpetration of the alleged crime. The chemical evidence against him was derived from an analysis of some of the bones of the deceased, in which arsenic was reported to be present by certain provincial practitioners, as well as by Orfila! (Journal de Chimie Médicale, Février 1847, p. 82.) If it were proved that arsenic existed naturally in bones, the discovery of it in this instance, could not be said to have furnished evidence of the *corpus delicti:* for by what intelligible rules could normal arsenic be distinguished from the minute portion taken into the system by absorption?

It is singular how long an error in Medical Jurisprudence, when once diffused, will continue to find circulation, although the experiments upon which it was based may have been long since refuted. The refutation of Orfila's opinion that arsenic existed as a natural constituent of *bone*, took place in 1841; but no case of poisoning by arsenic now comes to trial in which the most ingenious objections, founded upon his first experiments, are not urged to the chemical evidence of the presence of the poison. Rightly or wrongly, applicable or inapplicable, they are invariably raised by a counsel in defence. (See case of *Reg* v. *Richardson*, Med. Gaz. xxxvii. 919.) In one case, in which I was lately called upon to give evidence, where about five grains of solid arsenic was found lying on the mucous membrane of the stomach, Orfila's view that arsenic was a natural constituent of the tissues, was opposed to the chemical evidence. The inapplicability of the objection in this case, was immediately made evident by the judge asking the question, whether it was possible for a human being to generate spontaneously in the cavity and upon the surface of his stomach, five grains of solid arsenic! In the case of *Gilmour*, (Edinburgh, Jan. 1844,) Dr. Christison very properly said, in answer to an objection taken to his evidence on the detection of arsenic in the liver, "that it was no constituent part of the human body, and was not formed in it. The individual (Orfila) who first promulgated this theory, only argues now that small quantities are found in the bones; but in three several experiments before the Academy in Paris, he was unable to show it." Even if arsenic existed in bone, bones are never analysed. Notwithstanding this entire want of proof, the case of *The Queen* v. *Port*, (ante, p. 264,) shows that our judges are inclined to allow of its existence, because in this case, as it must necessarily happen with respect to absorbed arsenic, the quantity of poison detected was *small!*

Delicacy of Marsh's process.—Marsh's process is undoubtedly one of great delicacy. MM. Danger and Flandin assert that metallic deposits may be procured when the arsenic forms only the 2,000,000th part of the liquid examined. (De l'Arsenic, 83.) M. Signoret states that he has procured metallic deposits with only the 200,000,000th part of arsenic in the liquid: this is in the proportion of one grain of arsenic dissolved in about 400,000 ounces, or 3000 gallons of water! As the delicacy of this test has been already made a subject of discussion in a Court of law, (*The Queen* against *Hunter*, Liverpool Spring Assizes, 1843,) it may be proper to offer a few remarks respecting it. It was stated at that trial, that the one-millionth part of a grain of arsenic might be rendered visible by Marsh's test; and the judge, guided by this statement, put the question to another medical witness, whether arsenic could be so removed from the stomach in three days, as that it would be impossible to discover the one-millionth part of a grain in the body. It appears to me, that the facts relative to the delicacy of tests, are not always stated with sufficient clearness on these occasions. Thus we ought to know two points,—1. The total quantity of poison experimented on; and 2. The degree of dilution, or the total quantity of liquid in which the poison is dissolved or suspended. There is no doubt that considerably less than the millionth part of a grain of arsenic may, by Marsh's test, be rendered *visible* on a glass plate: it is possible to distinguish with the eye a piece of leaf-gold which would weigh less than the ten-millionth part of a grain; but the real question is, whether the test will discover arsenic in a single drop of solution, made by dissolving one grain of the poison in a million grains or sixteen gallons of water! If not, the statement amounts to nothing; for it is clear that if more than one drop of such an extremely diluted solution be taken, the test is acting upon a larger quantity of arsenic than the above form of expression would indicate. I have generally found that the fractional quantity stated to be detected, referred rather to the degree of *dilution* than to the *absolute quantity of poison* present: whereas a test may fail to act, as we have already seen, either from the smallest of the quantity of poison present, or from the very large quantity of water in which it is diffused. The results of my own experiments are, that where the arsenic is mixed with the acid liquid in a tube capable of holding two fluid ounces, very faint and scarcely perceptible deposits begin to be formed on a glass plate with a quantity equal to the 2160th of a grain: the diffusion here being equal to two million times the weight of the poison. With the 1080th of a grain in the same quantity of water, the arsenic forming therefore one-millionth part, slight brown annular stains were procured. The annular form is probably due to the central portion of the minute film being volatilized by the heat of the point of the flame:—unless the glass be speedily removed, the whole of the deposit may vanish. With the 720th of a grain, the arsenic being in the proportion of about the 600,000th of the liquid, the stains were much more decided, but quite imponderable. With the 100th grain in one fluid-ounce of water (the 48,000th part) and the 67th grain in two fluid ounces (the 64,800th part) the deposits on glass were decided and characteristic; and it is at this point, that the process begins to be safely available for the purposes of legal medicine.

Modifications of Marsh's process.—Many modifications of Marsh's process have been proposed. Thus MM. Danger and Flandin burn the gas in connexion with a cooled receiver, so that a solution of arsenic is thereby obtained. They make it the medium for extracting arsenic in a state fitted for testing. M. Lassaigne and Dr. Clark cause the arsenuretted hydrogen to pass into a solution of nitrate of silver, whereby arsenious acid (Lassaigne) is obtained in solution, and arsenuret of silver is precipitated. (Clark.) Berzelius, Liebig, and Köppelin and Kampmann, conduct the arsenuretted hydrogen through a tube instead of burning it; and the two latter chemists dry the gas by making it pass over fused chloride of calcium. The tube is then heated, and a clear ring of metallic arsenic becomes deposited at a little distance from the point to which the heat is applied. This result depends on the fact that arsenuretted hydrogen is easily decomposed, and its arsenic is then separated at a moderate heat without loss. All other metals which combine with hydrogen are deposited in the spot which is heated, and do not, like arsenic, form a well-defined ring in front of it. Mr. Morton proposes to produce the hydrogen by the galvanic decomposition of water, instead of by the action of sulphuric acid and water on zinc. In this way it is expected that we shall get rid of all the objections to the presence of arsenic or other impurities in the materials employed, the hydrogen evolved in this case being absolutely pure. Mr. Ellis has advised that the arsenuretted hydrogen instead of being burnt, should be decomposed by passing it over the dried oxide of copper,—the gas is absorbed without the aid of heat, and water, with probably arsenite of copper, results. Arsenious acid is then obtained by heating the oxide of copper in a tube. There are more inconveniences attending the use of some of these plans than are met with in the original apparatus of Marsh. M. Blondlot has made a useful suggestion for controlling the production of gas. He uses a Woolf's bottle with three openings, one for pouring in the acid, one for allowing the escape of the gas, and the third for a glass rod which is capable of being raised through the cork out of the acid liquid. On the lower end of this, a piece of pure zinc is rolled spirally, and by plunging it into or withdrawing it from the acid, the production of arsenuretted hydrogen is completely under command. (Comptes Rendus, 1845, ii. 32; and for a full account of the apparatus, see Journal de Chimie Méd. 1845, 616.)

REINSCH'S PROCESS.

In the application of this ingenious process, the solid or liquid suspected to contain arsenic, is boiled with about one-sixth part of pure muriatic acid, and a slip of bright copper foil or wire is introduced. If arsenic be present even in small quantity, the copper acquires either immediately or within a few minutes a dark iron-grey coating from the deposit of that metal. This is apt to scale off, if the arsenic be in large quantity, or if the liquid be long boiled. We remove the slip of copper, wash it in water, dry it, and gradually heat it in a reduction-tube, when arsenious acid will be sublimed in minute octohedral crystals: if these should not be apparent from one piece of copper, several may be successively introduced. A large surface of copper may be in this way at once covered—the grey deposit scraped off, and the powder gently heated in a reduction-tube. This test succeeds perfectly with powdered arsenic, the arsenites, arsenic acid, the arseniates, and orpiment. It will even separate the arsenic from the arsenite of copper, and from a common lead-shot. When the quantity of arsenic is small, the copper merely acquires a faint violet or blueish tint, and the deposit is materially affected by the quantity of water present, or, in other words, the degree of dilution. But one great advantage is, that we are not obliged to dilute the liquid in the experiment, and there is no loss of arsenic:—the whole may be removed and collected by the introduction of successive portions of copper. This process is extremely delicate, the results are very speedily obtained, and are highly satisfactory. One caution is to be observed, *i. e.* not to remove the copper from the liquid too soon. When the arsenic is in minute quantity, the deposit does not take place sometimes for a quarter of an hour. If the copper be kept in for an hour or longer, it may acquire a dingy tarnish merely from the action of the acid. This is known from arsenic by its want of metallic lustre, and its being easily removed by friction.

Objections to Reinsch's process.—Certain objections have been urged to this test. Thus arsenic may be present in the muriatic acid: this is at once answered by boiling the copper in a portion of the muriatic acid before adding the suspected liquid. This should always be a preliminary experiment. A more important objection is, that other metals are liable to be deposited on copper under similar circumstances. Thus this is the case with *Antimony*, whether in the state of chloride or of tartar emetic; nor is it always possible to distinguish by the appearance, the antimonial from the arsenical deposit. Should the quantity of antimony be small, the deposit is of a violet tint; if large, of an iron-grey colour, exactly like arsenic. *Tin* and *Lead* give a tarnish under the same circumstances, but there is no decided metallic deposit on the copper. *Bismuth* produces a deposit very closely resembling that of arsenic. With respect to *Mercury* and *Silver*, a metallic deposit takes place in each case without boiling. In a salt of nickel or cadmium, the copper undergoes no change; hence this is another important distinction between cadmium and arsenic. Lastly, if an alkaline sulphuret, or sulphuretted hydrogen, be present in the liquid, the surface of the copper will become tarnished; but this effect takes place on contact without boiling, and without rendering the addition of muriatic acid necessary. There is one answer to all these objections, namely, that from the arsenical deposit, octohedral crystals of arsenious acid may be procured by *slowly* heating the slip of copper or the grey deposit scraped from it in a reduction-tube. If this experiment be carefully performed, a ring of white arsenious acid will be easily obtained; this may be boiled in a few drops of water, and tested with the ammonio-nitrate of silver and sulphuretted hydrogen. Such a corroboration is necessary, because the crystalline form of arsenious acid is not always distinguishable; and the antimonial deposit gives a white amorphous sublimate, which, however, is quite insoluble in water. Care must be taken not to mistake minute spherules of water for detached crystals of arsenious acid. (See page 289.)

The facility of applying this process, renders it necessary that the experimentalist should be guarded in his inferences. It is not merely by the production of a stain on copper, that he judges of the presence of arsenic, but by the reconversion of the deposit causing the stain, to arsenious acid, demonstrable by its crystalline form or its chemical properties. If a deposit take place on copper, but arsenious acid cannot be obtained by heating it, then the evidence of its having been caused by arsenic is defective. Owing to the neglect of these corroborative results, antimony has been mistaken for arsenic. As tartarized antimony and James's powder are frequently employed in medicine, and may exist in the stomach at the time of death, it is highly necessary to guard against such a serious fallacy. If by heating the coated copper we procure a mixed sublimate of a doubtful nature, it is necessary to file off the ring of glass containing it, reduce it to powder, and boil it in a few drops of distilled water. The arsenious acid only becomes dissolved,—the oxide of antimony being insoluble in water. Unless from this solution we procure the reactions indicated with the ammonio-nitrate of silver, ammonio-sulphate of copper, and sulphuretted hydrogen gas, there can be no certainty that the deposit on copper was owing to the presence of arsenic. The larger the surface of copper presented in a small bulk, the greater will be the quantity of arsenious acid obtained. It was this which induced me two years since, to substitute for copper-foil the finest copper-gauze or woven-wire, containing sixteen thousand apertures to the square-inch. The surface here presented is exceedingly great. After the deposit has taken place, the gauze should be pressed between folds of blotting-paper, and then well-dried above the flame of a lamp. It may now be rolled into a small compass and introduced into a reduction-tube. To those who are not much accustomed to analysis, this plan may be more convenient than that of scraping the deposit from copper-foil and heating the powder. Of all the methods of detecting arsenic, there is none so simple, so speedy, or so easy of execution as this. Hence it has already in great part superseded most of the other more complex processes of testing for this poison.

Delicacy of Reinsch's process.—This test failed to detect the 4,000th part of a grain of arsenic in thirty drops of water, the dilution being equal to 120,000 times the weight of the arsenic. The deposit on copper commenced with a violet-coloured film, when the quantity of arsenious acid was equal to the 3,000th part of a grain in thirty drops of water, or under a dilution of 90,000 times its weight. It was also very decided with the 2,000th part of a grain in the same quantity of water, but in neither of these cases could octohedral crystals of arsenious acid be obtained by heating the copper. The following experiments will show how this test is liable to be affected by dilution:—the copper was coated in a few seconds, when boiled in a solution containing the 4,000th part of a grain in ten drops of water, although the test had failed to detect the same weight of arsenic in three times that quantity of water. So again, the 2,160th part of a grain in thirty drops of water gave an arsenical deposit on copper; while the same weight in half an ounce of water, did not produce any effect on the metal.

GALVANIC TEST.

If a small quantity of arsenic in solution, acidulated with muriatic acid, be placed in a platina capsule, and a piece of zinc foil introduced, a galvanic action ensues, by which metallic arsenic is deposited on the platina in a thin film. The capsule may be washed, dried, and gently heated. A plate of glass placed over it, will collect the arsenious acid, into which the metallic arsenic is converted by sublimation. I have found in this experiment, that the arsenic is deposited as much on the zinc as on the platina, and there is a great loss of arsenic from the production of arsenuretted hydrogen. On the whole, this is by no means an advisable mode of testing; and it is at the same time far inferior in delicacy to the process of Marsh and Reinsch. So much arsenic is occasionally deposited on the zinc, that by heating it slowly in a reduction-tube, octohedral crystals of arsenious acid may be readily procured from it.

Arsenic in liquids containing organic matter.—Arsenious acid, when in a state of solution, is not liable to be precipitated by any animal or vegetable principles, although all such substances render it less soluble in water. The liquid for analysis should be filtered through muslin, cotton, or paper, in order to separate any insoluble matters. Should it be coloured, this is of little moment, provided it be clear. If viscid, it should be diluted with water and boiled with a small quantity of muriatic acid; on standing, a deposit may take place, and this should be separated by a filter. As a trial-test, we may now boil in a portion of the liquid, strongly acidulated with pure muriatic acid, a slip of bright copper. In a few seconds, if arsenic be present, this will acquire a grey metallic coating. If, after half an hour, the copper remain unchanged, the arsenic, if present, must be in extremely minute proportion; if, on the other hand, the copper be covered by a grey deposit, it should be dried and heated in a reduction-tube in the way already described, in order to obtain from it octohedral crystals of arsenious acid. From several such slips of copper, or copper gauze, a quantity of metallic arsenic may be procured, sufficient, on reconversion to arsenious acid, to allow of a solution in water being made, to which all the liquid tests may be applied. One obstacle to the use of the gauze is, that oily and other kinds of animal matter, not easily removed by washing in water, may adhere to it. Digestion in ether and alcohol, slightly warmed, will free it from these substances, which are apt to become sublimed by heat, and obscure the result:—but it should be again washed in water and dried before heat is applied to it. When, however, much oily matter is present, it is better to boil the organic substance with muriatic acid, and filter the liquid through *a wet filter* before introducing the copper-gauze. In this way the fat and solid organic impurities may be separated. An even coating of arsenic was by this process obtained on copper-gauze from the decomposed tissue of the stomach of a person who had been buried nearly two years. As the gauze is remarkably hygrometric, it requires to be thoroughly dried in a vapour-bath before it is submitted to heat in a reduction-tube. Should there be any doubt whether the sublimate be caused by spherules of water or particles of arsenic, the tube itself should be kept some time in a vapour-bath. Water is dissipated at 212°. Arsenious acid requires a heat of nearly 370° for its sublimation.

By this process, the 144th part of a grain of arsenious acid was detected in two fluid-drachms of gruel, milk, porter, and other organic liquids, in so many different experiments. It was also thus easily separated from wine, brandy, the liquid contents of the stomach, the blood, and the tissues of the viscera. Here our analysis might be closed, if the object were to determine only the *presence* of arsenic, since a case can rarely occur in medico-legal practice, in which it would be necessary to extract the *whole* of the poison from the contents of the stomach or intestines.

Another process for procuring evidence of the presence of this poison in liquids, has consisted in transforming the arsenious acid to the state of sesquisulphuret, and in decomposing this compound by an alkaline flux. As a trial-test, we may first dip a piece of white filtering paper into the suspected liquid, and expose it to the action of a current of sulphuretted hydrogen gas in a tube. If arsenic be present, the paper will acquire a rich yellow colour, which immediately disappears on dipping it into a solution of ammonia. If the quantity of arsenic thus taken up by the paper, be less than the 4000th part of a grain, there will be no change of colour. We then test about half an ounce or an ounce of the liquid by passing the gas into it. Should the liquid for analysis contain oil, it may be separated in the way just described. Having satisfied ourselves that arsenic is present, we may get rid of a portion of the organic matter, by boiling the liquid with muriatic or acetic acid, and filtering. This object may be further accomplished by adding to the liquid when cold, one-third of its bulk of alcohol, again filtering and separating the alcohol by distillation. Sulphuretted hydrogen gas may now be freely passed into the liquid acidulated with either of the acids mentioned. When all further precipitation ceases, the liquid should be filtered, the precipitate collected, dissolved in ammonia, and precipitated by an acid. By digesting it in water, alcohol, and muriatic acid successively, it may be deprived of any organic matter combined with it, sufficiently to allow of its reduction by soda-flux or metallic silver in the way described. The sulphuret has sometimes a dark brown colour from adhering organic matter; it is then better to transform it to arsenic acid by boiling it in nitro-muriatic acid,—during which process, the organic matter is entirely destroyed, and a solution of arsenic acid is obtained and rendered fit for testing, by digesting the evaporated residue in distilled water; or the sulphuret may be deflagrated with nitre, and arseniate of potash then procured. In this case the surplus nitric acid should be driven off by sulphuric acid. An abundant deposit of metallic arsenic is obtained in either case by boiling the liquid, with muriatic acid and copper-gauze. In this way it is easy to analyse wine, coffee, tea, milk, porter, brandy, and similar liquids, for arsenic. This corroborative test is necessary, since I have known an instance in which a large quantity of orange-peel having been eaten and caused death, the contents of the stomach acquired a yellow colour from sulphuretted hydrogen gas, like that produced by arsenic. There was no deposit, and the yellow colour did not disappear on adding ammonia. (Page 23.)

Arsenic in oily compounds.—It has been already stated that arsenic is soluble in oil. The poison may require to be analysed either in this state or mechanically diffused in fat, butter, tallow, or similar substances. From all of these it may be separated by boiling them in a sufficient quantity of water, with about one-tenth of muriatic acid. The aqueous solution may then be freed from the oil by passing it through a filter previously saturated with water.

Contents of the stomach.—The contents of the stomach are often mixed with lumps of arsenic which may be separated by throwing those portions that do not pass through a filter into a large glass of distilled water, and after giving to it a circular motion, suddenly pouring off the supernatant liquid, when the heavy portions containing arsenic will be found at the bottom. The lumps may sometimes be left in the contents; they may then be easily removed, dried on filtering paper, and tested. If the arsenic has been taken in fine powder, there will be no lumps, but it will probably be deposited in masses, mixed with mucus and blood, on the coats of the organ in those parts where it is much inflamed and ulcerated. The arsenic in this state looks like moistened plaster of Paris, but it is of a darker colour, and when examined by a lens it is crystalline. It may be removed on a spatula, spread in masses on filtering paper, and slowly dried. As it dries, the granules will detach themselves from the mass, and they may be then easily tested either by the reduction or by Reinsch's process; *i. e.* by boiling the suspected particles, or even the stained portions of paper on which the organic matter has become dried, with muriatic acid and copper gauze. Mucus, blood, or even a layer of the mucous membrane of the stomach, may be thus easily tested. This is in general the only method which it is now necessary to employ. By the use of numerous tests and processes, a witness only exposes his evidence most unnecessarily to many ingenious objections. It is sufficient to obtain the deposit on copper: we then convert this by heat to arsenious acid,—which may be dissolved in water, and the silver, copper, and sulphuretted hydrogen tests applied to the solution with the usual results. In this way we avoid the troublesome and complex method of separating organic matter from arsenic. Care must be taken in examining a stomach not to confound pieces of bread or lumps of fat or adipocere with arsenic. Small portions of such substances appear very much like lumps of the poison. Bread is easily known by its being turned blue by iodine-water, and fat or adipocere is detected by laying the substance on a piece of white filtering-paper and passing beneath it a hot spatula—the fat melts and is absorbed by the paper: if the mass be arsenic, it will become simply dried.

Arsenic not always dissolved.—The fact that the liquid contents yield no arsenic, must not lead us to suppose that the poison is absent, I have found solid arsenic spread over the coats of the stomach in two cases, when the liquid contents yielded no traces of the poison in solution. In the same way I have detected no arsenic dissolved in tea when it was abundant in the sediment. (*Queen* v. *Lever*, 1845.) If none should be found either dissolved in the contents of the stomach or on the surface of the organ, we must cut off the inflamed and ulcerated portions of the mucous coat, and boil them with diluted muriatic acid and copper for half an hour. The liquid may be then filtered and tested. It often happens that no arsenic can be detected in the contents of the stomach or vomited matters, until after they have been boiled for at least one or two hours.

Arsenic not always present.—It is an important medico-legal fact, that in many undoubted instances of arsenical poisoning, not a trace of the poison can be found in the stomach or its contents. Several of these cases have occurred to my knowledge. In one, a girl took an ounce of the poison, and died in seventeen hours: there was much vomiting and purging, and the stomach-pump was used,—facts that might sufficiently account for the non-detection of poison in the body. In a second, nearly *two ounces* of arsenic were swallowed, and the person died in eight hours. No arsenic was discovered in the stomach. Even when there has been no vomiting and purging, the poison is not always found, but then the dose is generally small. Thus, in the case referred to me by Mr. Veasy, no arsenic could be detected in the stomach, duodenum, or their contents, although the patient had neither vomiting nor purging. Reasons have been already assigned for the non-discovery of the poison. (See ante, p. 114.) In cases of rapid death, however,—when the poison cannot be found in the *contents* of the viscera, it may always be discovered in the tissues.

Quantity of arsenic detected.—The value of the chemical evidence does not depend on the discovery of any particular *quantity* of poison in the stomach, —it is only necessary that the evidence of its presence should be clear and satisfactory. In the case of *Margaret Wishart*, Dr. Christison did not detect more than the one-fortieth of a grain of arsenic in the coats of the stomach; but this was deemed sufficient chemical evidence, and the prisoner was condemned and executed. (Edin. Med. and Surg. Journ, xxvi. 23.) Nevertheless there is a strong prejudice among lawyers that the chemical evidence is defective unless the quantity found be sufficient to cause death. (See ante, p. 117.) In the celebrated case of *Madame Laffarge*, Orfila's evidence was strongly objected to on this ground. He admitted that the quantity which he obtained from the body of the deceased was too small to be weighed, but estimated it roughly at half a miligramme, *i. e.* about the one hundred and thirtieth part of a grain!

Presence of arsenic in articles of food.—This poison is not likely to be met with as an accidental impurity in substances used for food. Some remarks will be made hereafter (p. 302) upon its alleged presence in corn which has been dressed with arsenic for agricultural purposes. So far as I have been able to ascertain, there is only one other case in which arsenic may be found in articles used for food—*i. e.* in *Vinegar*, as it is prepared by the decomposition of acetate of soda made from pyroligneous acid. M. Chevallier detected this impurity in some wood-vinegar which he was required to examine officially. The proportion of metallic arsenic found in it was the 1-2200th part, or less than seven grains to thirty-five ounces. The vinegar was, however, of that strength that it would require dilution with five or six parts of water before it would be employed for culinary or alimentary purposes: hence the quantity of arsenic present, was insufficient to occasion any injurious consequences, or to interfere with the inferences from a chemical analysis. The cause of the impurity was clearly traced to the sulphuric acid used in the distillation of the acetate of soda. This acid (obtained from pyrites) contained arsenic. Hence M. Chevallier recommends as a precaution, that no vinegar should be allowed to be sold as an article of food, except that which is prepared by fermentation. (Journal de Chimie, 1846, 330.)

Detection of absorbed arsenic in the tissues.—When arsenic cannot be detected in the contents of the viscera, it is necessary to adopt some method of extracting from the blood, secretions, muscles, or viscera of the deceased, that portion of the poison which has been *absorbed*. In most cases of acute poisoning arsenic will be found, but in variable quantities, in every one of the soft structures of the body—more abundantly in the viscera of the abdomen than elsewhere. In general the medical witness has it in his power to make a selection; but even here criminal ingenuity may be exerted to defeat his evidence. In a case tried in France in 1846, the body of a man named Glœckler, who was alleged to have died from poison administered by his wife, had been clandestinely removed, and thrown into the soil of a privy, where it was subsequently found. The abdominal cavity had been opened, and all the viscera removed, with the intention of obliterating all traces of the criminal act which had been perpetrated. The proofs of criminality rested entirely upon the chemical evidence, for the symptoms were not well-marked. It was clearly proved at the trial, that the wound in the abdomen had been made after death, and arsenic was readily extracted from the soft parts of the body in sufficient quantity to be weighed. The accused was convicted. (Gaz. Méd. Sept. 12, 1846, 726.) From this account it will be perceived that but for the newly discovered processes of detecting arsenic in the tissues, this crime must have passed undetected and unpunished. Except by the entire destruction of the body in a case of arsenical poisoning, a criminal cannot now defeat the objects of a chemical investigation. In the case of the *Queen* v. *Hunter*, tried at the Liverpool Spring Assizes, 1843, arsenic could not be detected in the contents of the viscera; and the judge suggested that the muscles or organs of the deceased should have been examined. This case shows that the detection of the absorbed arsenic must henceforth form a part of the duty of a medical jurist, when his other chemical experiments fail in discovering traces of the poison. There is no doubt that evidence of this kind, when carefully obtained, will be received by a Court of law. In the case of the *Queen* v. *Thomas*, tried at the Cardiff Summer Assizes, 1843, no arsenic was found in the contents of the stomach and intestines of one of the deceased persons; but the poison was detected in the liver by incineration with nitre. This evidence, although attacked in cross-examination on the ground (now refuted) that arsenic was a natural constituent of the body, was received as a satisfactory proof of the presence of the poison.

A case occurred not long since in France which clearly shows the great importance attached to this branch of the analysis. A woman was accused of having destroyed her husband by arsenic. The witnesses who had the investigation of the case, detected arsenic in the liquid contents of the stomach in well-marked quantity, but they omitted to extend their analysis to the tissues. The parties who were required to express an opinion of the cause of death from the medical facts, declared that the proofs of death from arsenic were insufficient, because, in order to justify this inference, the poison should be detected in the *tissues* of the body. The counsel in defence contended that the proof of poisoning had failed, and the jury acquitted the prisoner. (Gaz. Méd., 3 Janvier, 1846, p. 18.) It is very true that in *an unknown* case the presence of arsenic in the *contents* of the stomach may not be an absolute proof of death from this poison, unless there be at the same time some confirmatory evidence from symptoms and post-mortem appearances; yet, when these exist, the omission to examine the tissues cannot be fairly urged as an objection to the medical opinion of death from poison. Thus the tissues may contain no arsenic, for the deceased may have lived sufficiently long for its entire elimination. Nevertheless there are few cases in which this branch of the analysis should not be resorted to, although it entails much additional trouble. The detection of arsenic in the *tissues* make it clear that the poison must have been introduced during life, and that it has most probably caused death; its detection merely in the *contents* of the stomach or intestines, does not give this absolute proof. Besides, the poison found in these contents is not that which has caused death; this exists exclusively in the tissues of the organs. (See ante, p. 117.) It is worthy of remark that under the Medical Witnesses' Act (6 and 7 William IV. c. lxxxix,) the medical practitioner is only required to make "an analysis of the *contents* of the stomach or intestines," and for this only is a fee allowed!

The processes commonly employed for the discovery of arsenic in the tissues are those of Orfila, of MM. Danger and Flandin, and of Reinsch.

1. Orfila simply dries the animal matter and adopts the old process of incineration with nitre:—sulphuric acid is afterwards added to the lixivium, and the sulphate of potash produced, is separated by crystallization from the more soluble arsenite of that alkali, which is one of the products of incineration, if arsenic be present in the animal matter. The arseniate formed may be then analysed by Marsh's apparatus, but it would be preferable to employ Reinsch's process. In this way Orfila has detected arsenic in the blood, urine, and all the soft organs of poisoned subjects; but it appears that he was never able to discover the poison in less than eight ounces of blood and a larger proportion of muscular fibre; and he speaks of the occasional necessity of drying and incinerating the whole body! I have examined in this way four ounces of blood taken from one subject by two processes;—and the whole of the duodenum and part of the ilium of another,—both of whom had perished under the acute form of arsenical poisoning, but without any successful result. This process is by no means so satisfactory as the next to be described. Orfila has more recently proposed the use of chlorine, but the details have not been published.

2. MM. Danger and Flandin carbonize the animal matter by boiling it to dryness in a small quantity of strong sulphuric acid, about one-third the weight of the dried organic matter. They digest the resulting carbonaceous ash in nitro-muriatic acid, and after driving off the acid by a moderate heat, treat the residue with distilled water. This yields arsenic acid, if arsenic were present in the viscera, a compound easily discovered by Marsh's or Reinsch's process. In pursuing this process, I obtained from seven ounces of the liver of a man poisoned by arsenic, about a dozen minute sublimates, as well as the action of the vapour of the flame on ammonia-nitrate of silver. M. Blondlot has lately advised that the carbonization should not be carried to *dryness*, as it occasions a loss of arsenic; but when the liquid is of a pasty consistence, he passes into it a current of chlorine; the liquid is then filtered, and introduced into Marsh's apparatus, where it produces but little froth. (Comptus Rendus, 1845, ii. 32.)

3. The process of Reinsch is, however more easily applied than those just described; it is simply this. The soft organs (and for this purpose the liver is preferable,) are to be cut into very small pieces and boiled in a mixture of one part of

pure muriatic acid and eight of water, for two hours, or until the whole of the organic matter becomes a soft magma. The liquid may then be strained, and the residue pressed. If the quantity be large, it may be concentrated by evaporation. The copper gauze or foil should be next introduced, and half an hour or an hour allowed for the deposit if necessary. Should a deposit be formed, its nature must be positively determined in the way already described (p. 289.) I have had occasion to apply this process to the detection of absorbed arsenic in the tissues in many cases of arsenical poisoning with satisfactory results. A witness in making use of it, must always be prepared to meet with the following objection—namely, whether a deposit resembling that of arsenic may not be formed on the surface of copper by long boiling with animal matter (free from poison) and muriatic acid. Having tried on several occasions the liquid contents of the human stomach, the viscera, and even common muscle (beef,) as well as various articles of food, in order to determine this point,—the result has been, that except when arsenic was added, or when there was a very strong suspicion of its presence, no metallic deposit was formed on the copper. The metal came out of the vessel *untarnished*, or there was only a slight superficial discoloration (from oxide) easily removed by friction. It would be wrong however, to say, whatever suspicions may exist, that arsenic was present in any case, unless arsenious acid was obtained from the deposit. The analyst should remember that the liver, spleen, and kidneys, are the organs best fitted for yielding arsenic under these circumstances. The urine also contains it in the living and dead body.

4. Another plan for detecting poison in the tissues, consists in combining the processes of Flandin and Reinsch, *i. e.* by carbonizing the tissues with sulphuric acid, and then digesting the dry ash in muriatic acid and water. (For some additional remarks on these processes, see ante, p. 132.)

The detection of poison in the tissues has become of late years an indispensable part of the chemical evidence in those cases in which no poison can be discovered in the contents of the stomach and intestines. It is a fact of great interest to the medical jurist, that in whatever way arsenic may have penetrated into the body, it appears from the researches of Flandin to fix itself especially in the liver;—since about *nine-tenths* of the absorbed arsenic are found in this organ, the other tenth being unequally diffused through the other organs. (Op. cit. i. 562.) This gentleman does not consider that the proportion of arsenic present in the tissues, is always in a ratio to the quantity of blood which they receive. He was never able to detect arsenic in the brain, spinal marrow, or the bones. The lungs contained less than the abdominal viscera, the heart less than the lungs, and the muscles less than the heart. Other experimentalists have, however, arrived at different results. Dr. Brett informs me that, in 1846, he was required to examine the bodies of two children poisoned by arsenic, which had been buried four and seven weeks respectively. In one there was redness of the mucous membrane of the stomach, with fixed yellow patches in this organ and the intestines, appearing in places throughout the peritoneal coat. There was a bright yellow stain, probably from imbibition, on the thoracic surface of the diaphragm as well as on the surface of the spleen: these were not separable by washing. The stomach contained a mixture of arsenious acid and sesquisulphuret of arsenic. The tissues were examined by Reinsch's process after the organs had been well washed, with the following results. The diaphragm, lungs, and brain yielded arsenic; the pancreas more than the kidney; the lungs quite as much if not more than the liver. In the second child, arsenic was clearly detected in the tissues of the stomach and intestines, but equivocally in the liver. The boiling of the tissues in muriatic acid for the extraction of arsenic should take place in a *close vessel:* such as an alembic, or a capacious retort with a receiver attached to it. There is beyond all doubt a loss of arsenic when this precaution is not adopted. In May 1847, the following experiment was performed in order to determine this point. Four grains of arsenious acid were dissolved in five ounces of distilled water, to which five drachms of concentrated muriatic acid had been previously added. The vessel was so large, as to prevent the possibility of any liquid being carried over mechanically. The boiling was continued for two hours, when nearly one-half of the liquid had been collected in the receiver. This was found to contain arsenic on applying Reinsch's process. It has been long known that, in boiling saline solution, a portion of the salt is carried over. Thus when a solution of carbonate of soda is boiled, test-paper indicates that the vapour is alkaline.

It has been already stated (ante, p. 31,) that if an individual survive for a period of from *thirteen to fifteen days*, the absorbed arsenic will probably have entirely disappeared by elimination through the urine and secretions. An analysis may therefore fail to detect it. An important question on this subject arose in the French case of *Lacoste*. (Auch. Assizes, July 1844.) The deceased, who was alleged to have been poisoned by arsenic criminally administered to him, had for some time before his death employed a secret medicine containing arsenic, for the purpose of curing himself of an eruption of long standing; but he had ceased taking this medicine for *fifteen days* previous to his death. The prosecution relied strongly upon the fact, that the arsenic found in the various organs of the deceased, must have been administered to him since the period at which he had discontinued the use of the secret medicine. It was asserted, that within fifteen days the whole of the arsenic used medicinally, must have become eliminated. All the medical witnesses agreed that after fourteen days no absorbed arsenic would remain in the body; and M. Flandin, who has performed many experiments on this subject, considers this the extreme limit for the human subject. (Des Poisons, i. 739.) His experiments on animals gave different results: in young and healthy dogs the elimination was complete in from six to ten days; in sheep, thirty days. The different physiological conditions of these animals, render these results inapplicable to the human subject. This medical inference, in the case of *Lacoste*, was strongly disputed by M. Bonjean of Chambery. He mentions a case in which a patient took medicinally arseniate of soda, prescribed for him by Dr. Cazenave. In twenty-four days he took, in divided doses, only *three-quarters* of a grain of arseniate of soda, equal to about half a grain of arsenic acid. A *month* after he had ceased taking the arsenical medicine, M. Bonjean examined a pound of his urine, and from this, by the aid of Marsh's apparatus, metallic arsenic was obtained. M. Bonjean considers that absorption is more complete with small doses than with large; but this does not remove the difficulty, which is to explain why, when a substance is more rapidly absorbed, it should not be more rapidly eliminated. An account of the case of *Lacoste* will be found in Flandin. (Des Poisons, i. 637; see also Ann. d'Hyg. 1846, ii. 155; and Monthly Journ. Med. Science, Dec. 1845, 933.) It is extremely doubtful whether such speculative opinions would have been received as evidence in an English court of law. It is very likely that, in most cases, arsenic is eliminated within fifteen days; but should traces of the poison (unabsorbed) remain about the alimentary canal, it may continue to be eliminated for a much longer period. Observations are neither sufficiently numerous nor accurate, to justify our fixing so close a limit.

Arsenic discovered in the dead body after long periods.—When the poison is really present in the stomach at the time of death, it does not easily disappear, and it may therefore be discovered for a long period after interment. White arsenic slowly becomes changed to yellow sesquisulphuret by the evolution of sulphuretted hydrogen in the decomposition of the stomach or its contents. It forms then a deep yellow stain on the surface, appearing like mustard. I have thus found it converted to sulphuret twenty-eight days after interment (*Reg.* v. *Jennings*, Berks Lent Assizes, 1845:) but this change may take place in a much shorter period. In recent cases the conversion is, however, in general only partial, as white grains may be often seen in the yellow mass. (See post, p. 378.) In *Mrs. Smith's* case, the sulphuret of arsenic was discovered in the stomach fourteen months after interment. In the cases of the *Cheshams* (Essex Lent Ass. 1847,) which I was lately required to examine, the coats of the stomachs were in both instances deeply dyed with large patches of yellow sulphuret nineteen months after interment. Arsenic has been detected in the body at the end of three years (Galtier, Toxicologie, i. 370,) and even after seven years. (Devergie, i. 313.) Wöhler is stated to have detected it in the bodies of two men seven years and six months after burial; this, however, was absorbed arsenic, and the process pursued was incineration of the soft organs by nitre. (Ann. der Chim. und Pharm. liii. 141; Chemical Gazette, 1845, 192; Med. Gaz. xxxv. 655.) The longest period at which it is reported to have been detected was *ten years*. The body had in this case become reduced to a skeleton. A confession was made by some of the parties concerned in the murder: corroborative evidence was sought for, and as it was asserted that a large dose of arsenic had been given, and the person had died in twenty-four hours, the remains of the skeleton, identified as that of the deceased, were submitted to chemical examination, when arsenic was readily detected. The examination of another skeleton found near that of the deceased led to a negative result: no arsenic was found. The jury were satisfied with this evidence, and returned a verdict of guilty. (Journal de Chimie Méd., Fevrier 1847, 82.) The particulars of the analysis are not given, nor is it stated whether the earth around the skeleton did or did not contain arsenic. When the contents of the stomach are not allowed to drain away, the poison may be detected after a very considerable period. A person died from the effects of arsenic on the 21st of February, 1834,—the poison was at the time easily found in the contents of the stomach; these have now been kept for upwards of *thirteen* years loosely covered, and arsenic is still as readily to be detected in them as in the first instance, whether Marsh's or Reinsch's process, or sulphuretted hydrogen, be employed.

Inference of death from arsenic when the poison is discovered at long periods after interment.—When arsenic is discovered in the remains of persons who have died many months or years previously, it may be a question how far a medical witness is entitled to infer that death had taken place from poison. I have elsewhere made some remarks in reference to one branch of this inquiry (p. 61,) where it is assumed that something is known concerning symptoms and post-mortem appearances, and the case is recent. It then becomes a simple question of diagnosis; for it is undoubtedly true that a man who has swallowed poison which requires some hours for its fatal operation, may die of latent disease of a suddenly fatal kind. But the case is different when we are examining a body eighteen months or two years after death. As an abstract proposition, the discovery of arsenic, even in large quantity, in such a case, would not, *per se*, prove death by poison. It would, however, furnish strong presumptive evidence that arsenic was the cause of death, especially if any of the poison (absorbed arsenic) were found in the tissues of the body. This question was put to me at the trial of the *Cheshams* (Essex Lent Assizes, 1847,) just mentioned. In the stomach of one child, there were found about ten grains, and in the stomach of the other, about twenty grains of the yellow sulphuret of arsenic. The mucous membrane beneath was of a dark red colour, and the coats, which contained in every part an abundance of absorbed arsenic, were, on the whole, well preserved. I did not examine the rest of the body, and had the examination been made, no physical cause of sudden death operating eighteen months before, would have been detected. Evidence was given to show, that one child died in about twenty-four hours after the commencement of its illness, and that it had laboured under continual vomiting and intense thirst. Under these circumstances, I did not hesitate to state that, in my opinion, the deceased child had undoubtedly died from the effects of poison; and no attempt was made to invalidate this opinion on the part of the defence. The learned judge who tried the case (Lord Denman) subsequently suggested to me that such an inference from the discovery of poison, could not be fairly drawn in all cases. Some corroborative evidence would undoubtedly be required when the poison was slow in its operation; and this would be more especially necessary, when the soft parts were entirely decomposed, and the arsenic was found mixed with disintegrated portions of the skeleton.

Arsenic in the soil of cemeteries.—It appears from the researches of several toxicologists, that the soil of grave-yards often contains a compound of arsenic, generally in an insoluble form. In eight trials on four different soils, Orfila found three of them arsenical. He used about six pounds of earth in the experiment. As there was no sign of arsenic, except when an acid was used, he inferred that it existed in the state of arsenite or arseniate of lime. The researches of Flandin have corroborated this result; and, in one instance, this experimentalist estimated that the quantity of arsenic, in an insoluble form, in about a pound of earth, did not exceed the twentieth part of a grain! Admitting the existence of arsenic as a natural constituent of certain soils, it becomes important to determine how far it may affect the chemical evidence of the presence of this poison in the remains of bodies which have undergone exhumation. If the coffin be cracked or entirely destroyed, so that the earth has become intermixed with the remains, and that which surrounds the coffin yields traces of arsenic, it is evident that no reliance could be placed upon the inference that the arsenic existed in the dead body, unless the poison found in the remains was in extremely large proportion. The reader will find cases in which doubts based upon the origin of the arsenic found in the decomposed dead body, led to the abandonment of chemical evidence. (Flandin, Traité des Poisons, 674, 683.) A difficulty of this kind cannot, however, when proper precautions are taken, often present itself in practice. A body buried in a coffin is rarely so far decomposed as to become covered by the soil from the disintegration of the coffin in a period shorter than from seven to ten years; and until such a complete disintegration has taken place, it is not easy to perceive how the presence of an *insoluble* arsenical compound, as a natural constituent of the soil, can present any objection to the results of an analysis. In the examination of these soils, it has been ascertained that no arsenical compound *soluble in water* has existed in them; therefore, if distilled water should yield on boiling the remains, a solution of arsenic, the presumption is that it could not have been derived from the soil. It has been supposed that the arsenic may have been carried by percolation from the soil into the body: but in this case, as Flandin has observed, the exterior of the body would contain more than the interior: while in a case of arsenical poisoning (except when dependent on local application) the liver and stomach would yield more than the skin. (See Galtier, i. 368; Flandin, i. 429, 741.) M. Devergie thinks that a body buried without a coffin, and covered simply by a shroud, might thus, under the access of water, imbibe arsenic from the soil: and such is the opinion of M. Vanden Broeck, even when the compound of arsenic is perfectly insoluble in boiling water. (Flandin, i. 442.) This opinion is, however, directly opposed to observed facts.

Is the arsenic in a soluble form?—This question has acquired considerable importance from the result of the trial of *Elizabeth Johnson* for poisoning her husband with arsenic. (Liverpool Lent Assizes, 1847.) The accused, concerning whose guilt, morally speaking, there could be but little doubt, appears to have owed her acquittal entirely to the assumption that arsenic in a *soluble* form, might have found its way into a dead body through a crack in a coffin, although it had not been shown that the soil of the churchyard where the body was buried contained any trace of the poison, either soluble or insoluble! Still, so far was this scientific question carried, that the assumption of arsenic being present in the soil, was allowed in favour of the prisoner. The deceased died on the 3d of December, 1846: the evidence that he had died from arsenic, was not then rendered complete by the post-mortem examination and analysis. The body was exhumed on the 9th of March, 1847; and Mr. Watson, of Bolton, to whom I am indebted for the particulars of the case, examined the viscera. This gentleman clearly detected arsenic in the liver, intestines, heart and blood, kidneys, gullet, and tongue, and in the muscular substance of the right thigh. The largest proportion of poison was derived from the liver, kidney and intestines; and the smallest quantity, amounting to only an exceedingly slight trace, was detected in the heart and blood. Mr. Watsondid not hear the medical evidence given at the trial, in consequence of his having been ordered out of court with the other witnesses: but it appears that Mr. Leigh, the chief medical witness, who was present at the exhumation of the body, stated in his evidence that there was a large quantity of "fluid blood" in the body. When asked by the judge to account for this, he said he thought it might have arisen from the penetration of water, as the grave was wet, and the coffin was split from one end to the other. The body had only been buried three months, but the wood was very thin, and had apparently cracked from the superincumbent weight of earth. The judge then asked, "Supposing there to be arsenic in the soil of the churchyard, was it not possible for some of that arsenic to be washed into or introduced into the body along with the water?"—to which Mr. Leigh replied, he thought it was possible. The counsel for the prosecution, not knowing probably how to frame his scientific questions so as to have, after this statement, a proper and clear explanation from Mr. Watson as to how far the arsenic found in the body was or was not likely to be due to the assumed presence of soluble arsenic in the soil, and its introduction into the body by water;—and Mr. Watson, not knowing what an important point respecting his chemical evidence had been thus raised during his compulsory exclusion from the court,—this very material statement was allowed to pass to the jury as not only a possible but a probable mode of accounting for the presence of arsenic, not in the blood only, but in the liver, the kidney, intestines, and even *the tongue, œsophagus, and muscles of the thigh!* The first intimation which Mr. Watson received of the mode in which his chemical evidence would be applied, was in the charge of the learned judge to the jury: and there can be no doubt that from arsenic not being clearly detected in December, the jury referred its discovery in the dead body in March, to the series of assumptions above detailed. As the *smallest* quantity of arsenic was found in the *blood*, which it was assumed was mixed with water (the supposed actual solvent of the poison,) it is utterly impossible to refer the presence of the arsenic in the other viscera of the body to the action of water,—even admitting, what is contrary to all experience, that the soil of the church-yard contained arsenic in a *soluble* form. The liver and kidney contained the largest, the heart and blood the *smallest* portion of poison—only a *minute trace*, as I am informed by Mr. Watson. There was even more arsenic in the tongue and gullet and muscles of the thigh respectively than in the heart and blood, *i. e.* than in the very medium from which the whole body was supposed and considered by the learned judge and jury to have derived its poisonous impregnation in the short period of three months from a crack in the coffin lid!

This case furnishes some important subjects for reflection:—1. Medical witnesses, who are required to give evidence on intricate points of science, should always be allowed to be present in court, if the object of a criminal trial for poisoning be to elicit the *whole* truth! 2. In conducting an analysis in the first instance, it should be so complete, even when suspicion is weak, that there can be no further ground for the subsequent exhumation of the body, and the chemical analysis of the viscera. 3. A portion of the soil near the coffin, in a case of exhumation, should always be examined for arsenic, by the process to be presently described. 4. It would be advisable to examine not only the muscles, but also the *skin* immediately over the muscles. In the event of the introduction of arsenic by percolation, the skin should contain much more of the poison than the muscles beneath it. 5. It is always important, in exhumations, to notice the relative proportion of arsenic in equal weights of the different viscera or parts of the body examined. This was here done by Mr. Watson, and it was unfortunate for the cause of justice, that the proper steps were not taken by the court to arrive at the truth. That arsenic is not thus washed into a dead body buried in an arsenical soil, is not a mere speculative opinion,—it is based on fact. In 1844, M. Ollivier made the following communication to the French Academy. The body of a female, alleged to have been poisoned by her husband, was exhumed, and it was ascertained that no poison was present, and that she had died from natural causes. A married woman, who wished to marry this man, poisoned her husband, and arsenic was detected in his body (the liver,) as well as in the earth of the cemetery in which it was buried. From a suspicion that the analysis had not been carefully made, the body of the female, which had been buried in the same cemetery, and which it appears, on reinterment, had accidentally fallen out of the coffin into the grave, and had become completely covered by the arsenical earth, was again exhumed after several months had elapsed. Not a trace of arsenic, however, was detected in the body: hence it appears evident that, under the most favourable circumstances, the human body does not acquire an impregnation of arsenic from contact with arsenical earth. An abstract of this case appeared in an English journal some years since. (Lancet, Aug. 17, 1844, 638.) Hence the mode in which medical evidence was dealt with at the trial of *Johnson* was not only in violation of all probability, but directly opposed to ascertained facts!

Another recent case, in which this question respecting the presence of arsenic as a constituent of the earth of cemeteries arose, was that of the *Queen* v. *Richardson*. (Med. Gaz. xxxvii. 919.) Its presence in the earth was here clearly negatived by the medical witness, who had taken care to make an analysis of it.

Analysis.—In the analysis of the *soil*, boiling water should be first employed as a solvent; and if this should take up no arsenic, we may then use one of the mineral acids; for this purpose pure muriatic acid, diluted with from six to eight parts of water, may be employed. Iron and lime will be chiefly dissolved, and these may be got rid of by supersaturating the acid liquid with bicarbonate of potash, and filtering. The filtered liquid, acidulated with muriatic acid and boiled with copper, will yield, if the poison be present, an arsenical deposit. This deposit should be converted to arsenious acid by oxidation, and tested with the liquid tests, before it is pronounced to be arsenic. The use of Marsh's test is liable to many fallacies. In the series of Norfolk poisonings, the investigation of which was conducted by Mr. Firth, in the summer of 1846, it was proved that the soil of Happisburgh churchyard, in which six of the bodies were buried, contained arsenic, although taken at the distance of *three feet* from the graves of the poisoned family! Mr. Firth informs me that the poison was easily detected in *half* an ounce of the earth by the process of Reinsch. He could not procure any trace of the poison by acting on the soil with boiling water, but readily by using muriatic acid as a solvent. One of the bodies had been buried eleven years; therefore this discovery of arsenic in the soil was really of practical importance. A mass of the decomposed remains was scraped up with a spoon from the sides of the lumbar vertebræ; and arsenic was found by Reinsch's process, but in very unequal quantity in equal parts of the remains. Mr. Firth, knowing that the soil of the churchyard contained arsenic, referred the poison to this source, not to any introduced into the body of the deceased during life. This was a very proper precaution, notwithstanding the suspicion of death from poison in the case of the deceased. The observations of Mr. Firth on the insoluble state of the arsenic in the earth around the decomposed remains of the deceased, appear to furnish an answer to an ingenious speculation which has been lately advanced, namely, that ammonia, generated by putrefaction, might act as a solvent to the arsenical compound, and, under the percolation of water, convey the poison into the dead body. (Gaz. Méd. 12 Juin, 1847, 452.) It is only in the latter stages of decomposition that the body is likely to be so exposed to the soil, as to render it possible for the cemetery-arsenic to be transferred to it; but then the production of ammonia ceased, for all the soft parts will have become destroyed. It is not unlikely that arsenic may be slowly eliminated from the dead tissues by the production of hydrosulphuret of ammonia, or even by the formation of arsenuretted hydrogen; but so little influence has this change, that the speedy conversion of the poison to sulphuret tends to fix it permanently in yellow patches in the substance of the organs. I have thus discovered it in large quantity nearly two years after interment, in a stomach abounding with hydrosulphuret of ammonia. Orfila has, however, very recently put this hypothesis to the test. He procured a large quantity of earth taken from the cemetery of Epinal, which was known to be impregnated with arsenic. He buried in this earth a full-grown fœtus, the liver of an adult, and various portions of dead human bodies. *Three months* afterwards these various parts were exhumed, and were found to be in a complete state of putrefaction. They were carefully examined for arsenic by the usual processes, but not a trace of the poison could be detected. (Acad. of Med. 29th June, 1847; also Gaz. Méd. 3d July, 1847, 535.) There is therefore no ground, either in fact or theory, for the assumption that insoluble arsenical compound of the earth of a cemetery is rendered soluble by the generation of ammonia from putrefaction, or that it is ever thus washed into the dead body. These experiments render it still more apparent that, in the case of the *Queen* v. *Johnson* (ante, p. 297,) the acquittal took place upon a chemical mistake.

There are many speculations as to the source of the arsenic in the soil of cemeteries. Orfila at first referred it to the disintegration of human bones; but as it was clearly proved that no arsenic was naturally contained in them, there was at once an end to this hypothesis. From the late researches of M. Walchner, it would appear that arsenic as well as copper is a constituent of all soils abounding in oxide of iron, and in all the ochreous deposits of acidulous waters. It is also naturally contained in some mineral waters under the form of arseniate of lime. He has thus discovered it by the employment of muriatic acid in all kinds of clay, marl, or earthy deposits coloured by oxide of iron, and he believes it to be universally diffused. (Comptes Rendus, Sept. 21, 1846, 612.) On this view it may exist in other localities as well as in the soil of churchyards; and would only be found in the latter when the earth was of a highly ferruginous character. Most kinds of pyrites contain arsenic in the state of mispickel. Arsenical pyrites contain nearly forty-six per cent.; the remainder consisting of sulphur and iron. Iron pyrites abound in the strata on the surface of the earth, especially in chalk, clay, and marl; and it is not improbable that, by the slow decomposition of this substance, oxide of iron, sulphate of iron, and arseniate may result. This may become diffused through the soil and render it arsenical.

It does not appear to me probable that arsenic is a frequent constituent of the soil even in an insoluble form; and when it occurs, the proportion will probably be so small, that an enormous quantity of earth will commonly be required, in order to demonstrate its presence. In July 1847, I examined a pound of the earth taken from a London cemetery, which had been used for burials for several centuries. The earth was procured from the bottom of a grave eight feet in depth, and from a spot where it was mixed up with disintegrated bones and portions of old coffins. It was sifted to separate the pebbles and loose bones, boiled for two hours in a quart of water, and the liquid filtered. This was concentrated to three ounces. Marsh's and Reinsch's processes were applied to equal portions of the concentrated liquid, but not a particle of arsenic existed in it. On adding a minute portion of arsenic (about 1-3000th part,) the poison was immediately indicated by these two processes. The residuary earth, insoluble in water, was then boiled with three ounces of pure muriatic acid and twenty-four ounces of water, for two hours, and the acid liquid was filtered and concentrated to four ounces. A portion was supersaturated by bicarbonate of potash, and the whole of the lime and iron separated. On trying the two processes above mentioned, there was not the slightest indication of the presence of arsenic. The evaporated acid liquid was also tried, but no arsenic was found. The acid had merely dissolved an enormous quantity of lime, some oxide of iron, and traces of magnesia. In employing such a liquid in Marsh's apparatus, it is necessary in the first instance to precipitate the lime by sulphuric acid.

An interesting case was tried in May 1846, before the Court of Assizes of Herault in France, in which the question relative to the presence of arsenic in the soil was material. Three persons were charged with the murder of a man by poison, in August 1844. The principal symptoms were cold sweats, irritation in the extremities, and emaciation. As this was a case of *slow* poisoning, and the disorder had intermission, no suspicion arose until it was found that the symptoms always became aggravated when his wife (one of the prisoners) came to visit him. He was now seized with vomiting, and his wife was observed to collect carefully, and throw the vomited and other matters out of the window of the room. One day the deceased happened to vomit on the floor, and the wife was observed to cover the spot with ashes, and afterwards, to wash it herself with potash. The husband lingered and died on the 6th December, the wife being his only attendant; and she stated to her neighbours that he went off gently and without convulsions. Some suspicion arose,—the body was exhumed, and an inspection was ordered; but putrefaction had already commenced. The viscera of the abdomen were, however, found to be well preserved. Arsenic was discovered in the tissues and contents of all the viscera of the abdomen and thorax.

The medical witnesses having been informed that the wife had thrown the vomited matters out of a particular window, they proceeded to collect the earth below for some space around. The floor of the chamber was scraped, until sufficient material was collected for analysis. Arsenic was distinctly discovered in the earth by the wall, immediately under the window; and in smaller quantity for a circuit of more than two yards. Beyond this space the earth was not found to contain any traces of arsenic. The scrapings of the floor, notwithstanding the washings with potash, gave traces of arsenic in the parts where the vomited matters had fallen, but not in any other spot. MM. Audouard and Bernard, from these results, gave an opinion that the deceased had died from arsenic. In order to obviate any objection that this poison might have penetrated into the body from the soil of the churchyard where it was buried, a portion of this was collected and examined, and was found to be free from arsenic. It was urged that the arsenic was a normal constituent of the body; but this was distinctly denied on the grounds already stated (p. 284.) The wife was convicted of the murder. (Gaz. Méd. 20 Juin, 1846, 498.)

A singular question arose, on the trial of *Laffarge*, in reference to the presence of arsenic in iron. The quantity of absorbed arsenic extracted from the body of the deceased did not exceed the *one hundred and thirtieth part of a grain!* (·0077 gr.) The deceased Laffarge having been a smelter of iron, it was ingeniously suggested in the defence that this small portion of arsenic might have been absorbed into his body in a state of vapour during his attendance at his forges, and thus account for the minute portion of poison detected by Orfila. It turned out, however, that deceased had not been near the forges for a month before the fatal symptoms appeared; therefore, as the effects were not likely to remain dormant, the poison could not be referred to this source. On the assumption that, according to the researches of M. Walchner, the oxides of iron always contain arsenic, it has been supposed that the poison might find its way into the body by the employment of the hydrated oxide of this metal as an antidote in cases of arsenical poisoning. An attempt was also made to account for the arsenic found in the body of Laffarge on this ground. I have examined several specimens of the artificial oxides of iron by the processes of Marsh and Reinsch, without detecting any trace of arsenic. This exception to chemical evidence appears to me to be inadmissible. In *Laffarge's* case, a much better objection would have been that the employment of large quantities of nitric acid and nitre in an iron vessel in stewing down the whole body of the deceased, might have accounted for the minute fractional quantity of arsenic detected.

Upon more plausible grounds it may be contended that minute portions of arsenic would find their way into the body through that universal article of food, —bread. Seed-corn is often soaked in a solution of arsenious acid in order to destroy the spores of the fungi producing smut: it might be argued that some of the poison would exist in the crop, and when eaten as bread might slowly impregnate the system. This question has not escaped the notice of chemists. M. Audouard states that he has detected arsenic in the crop of corn when the seeds had been previously soaked in a solution of arsenious acid. The poison was, however, in very minute quantity. On the other hand, M. Girardin, by a satisfactory series of experiments, has proved that there is no detectable quantity of arsenic in corn under the circumstances. In some of his experiments he used more than four pounds of corn, and he could not discover in this large quantity, the least trace of arsenic. (Annuaire de Chimie, 1846, 686.) This question must therefore be considered as settled in the negative. Independently of these facts, it must be remembered that arsenic is not an accumulative poison, that in minute quantities it is eliminated as fast as it is received (p. 259, ante.)

Arsenic in solids.—Arsenic may exist in solid articles of food, such as bread, pills, and powders:—in ointments, and certain kinds of candles;—or matters vomited by a person poisoned, may sometimes be imbibed by articles of clothing or furniture. In all these cases we should simply boil the solid in water, with the addition of muriatic acid and copper; or if we wish to separate the whole of the poison, we may proceed, as in the case of organic liquids, by using a current of sulphuretted hydrogen gas. A cat was poisoned by half a drachm of arsenic—the animal died in about nine hours. No trace of poison was found in the body; but a small part of the floor of the room, where the cat had vomited, was scraped off, boiled in water, and yielded on analysis, clear evidence of the presence of arsenic.

QUANTITATIVE ANALYSIS.

The whole of the arsenic from a measured portion of the liquid must be precipitated by sulphuretted hydrogen, as sesquisulphuret. This should be purified by dissolving it in ammonia, and reprecipitating it by muriatic acid: it may be then washed, dried and weighed. Every *hundred* grains of sesquisulphuret obtained, indicate about *eighty* grains of white arsenic (100 : 80·4.) The quantity may be thus determined by multiplying the precipitate by 4, and dividing the product by 5. Should the sesquisulphuret be very impure, it may be converted to arsenic acid, and the proportion calculated by precipitating this as arseniate of silver. It has been recommended, in order to determine the quantity of arsenic contained in the tissues, to connect Marsh's apparatus with a horizontal glass tube, secured by a stop-cock, and dipping at a right angle into a solution of nitrate of silver. Heat being applied to the horizontal tube, the metallic arsenic is deposited in a succession of rings, and what escapes deposition, is decomposed and collected by the nitrate of silver. The quantity of arsenic is then estimated, and compared with the weight of organic matter employed. This plan is much too complex for general employment. It has been also stated that Reinsch's process would serve to indicate the quantity of arsenic; but this statement must have arisen from imperfect theoretical notions. If the copper be weighed before and after the deposit of arsenic upon it, the increase of weight in the latter case does not indicate the quantity of arsenic; because a portion of the copper is invariably lost as chloride,—a fact proved by adding ammonia to the liquid. If the copper be weighed with the arsenical deposit, and afterwards heated to expel the arsenic,—the loss of weight does not indicate the quantity of arsenic; because in being heated, the copper becomes partially oxidized—a fact apparent on inspection. The only satisfactory plan, therefore, to determine the quantity of poison, is to obtain the arsenic in the state of dry sesquisulphuret.

[An abstract of cases of poisoning by Arsenic will be found in Beck, and a copious bibliography is appended to the article Arsenic in Amer. Cyclop. Prac. Med.—In the case of Mina a fact given in evidence by several of the medical witnesses, is of interest. They stated that when the stomach of Mr. Chapman was opened, it give out a smell of red herrings. Dr. J. K. Mitchell also says that a stomach of a man in which he placed some arsenite of potash, acquired the same odour. In a similar experiment with arsenious acid I obtained an analogous result.—G.]

ARSENITE OF POTASH.

The compounds formed by arsenious acid with the alkaline bases are all poisonous. Those of potash, soda, and ammonia, are soluble in water, and therefore, act with more energy. The **Arsenite of Potash** is the only preparation which here requires notice. It is used in medicine, and is well-known under the name of **Fowler's Mineral Solution**, or Tasteless Ague Drop. It is made by boiling arsenious acid with carbonate of potash, the latter being in slight excess, and it is coloured with compound tincture of lavender. In the preparation of the London Pharmacopœia, there are sixteen grains of arsenious acid in thirty-five fluid-drachms of the solution, which is nearly equal to one grain in 2.06 fluid-drachms. Its real strength may be affected by any impurities in the arsenious acid employed. The preparation used in Scotland is of the same strength; but that of the Dublin College is one-ninth weaker.

The action of this liquid as a poison, in large doses, is in all respects analogous to that of arsenious acid. The medical dose is from four to thirty minims twice a day. It is common to commence with four to five minims, and gradually increase the dose. Dr. Pereira has known fifteen minims to have been taken three times a day for a week without ill effects; and Dr. Mitchell, of Ohio, has given from fifteen to twenty drops three times a day in intermittents. (Materia Medica, i. 649.) In some persons there is a strong idiosyncrasy with respect to arsenic (see ante, p. 268;) and even smaller doses than those commonly prescribed, can hardly be borne without causing alarming symptoms. A dose of from two to three drachms would probably suffice to destroy the life of an adult. A case was reported, in the Pharmaceutical Journal for 1845, in which one drachm (equal to half a grain of arsenic) was taken with comparative impunity. I have not met with any recorded instance of death from poisoning by this compound. Orfila refers to a singular case of poisoning by a compound arsenite of potash and lime in a solid form (*i. e.* as a soap,) in which the most marked nervous symptoms (trismus) appeared in three-quarters of an hour: the individual recovered. (Toxicologie, i. 449.)

The TREATMENT of a case of poisoning by a soluble alkaline arsenite would be the same as that for arsenious acid; but the hydrated sesquioxide of iron might be given with a greater prospect of benefit.

ANALYSIS:—This solution has the odour of tincture of lavender, is of a reddish colour, and has an alkaline reaction. It gives at once a green precipitate (arsenite of copper) with the sulphate of copper, and a yellow precipitate with nitrate of silver. Acidulated with muriatic acid, and treated with a current of sulphuretted hydrogen gas, it yields a yellow sulphuret; and when boiled with muriatic acid and copper, a deposit is obtained which readily furnishes octohedral crystals of arsenious acid. (See REINSCH'S PROCESS, ante, p. 286.)

For an account of poisoning by ARSENITE OF COPPER, see post, SALTS OF COPPER.

METALLIC ARSENIC. FLY-POWDER.

It is generally considered that metallic arsenic is not poisonous; but, as it is very easily oxidized, it speedily acquires poisonous properties. According to Berzelius, the metal is slowly converted, by exposure to air, to a pulverulent suboxide of a black or brownish-black colour. This is commonly called Fly-Powder, a name also applied to the arsenical cobalt ores reduced to powder. Thus, what is called the Tunaberg ore, a mixture of cobalt, arsenic, iron, and sulphur, is largely used on the continent under the name of Fly-Powder: and, as it comes within the reach of children, it frequently gives rise to accidents. A few years ago, Dr. Schobbens was called to a man who had swallowed some by mistake for a purgative. He was soon attacked with the usual symptoms of poisoning by arsenic. He swallowed a large quantity of milk, which occasioned immediate vomiting. As fifteen hours had elapsed before a medical man saw him, no treatment was of any avail, and he died from the effects of the poison. In another case, a child, aged four years, swallowed a portion of fly-powder. The hydrated sesquioxide of iron was given every half hour, and the child recovered the next day. (Monthly Jour. Med. Science, Sept. 1846, p. 228.) The exact quantity taken in these cases is not known; but there is no doubt that the poison is but little inferior to arsenious acid in activity. The symptoms and post-mortem appearances from a fatal dose would be identical. This substance is not much known in England. A woman was convicted in France for poisoning her husband with it in 1844. (Briand, Man. Comp. de Méd. Lég. 452.) It owes its poisonous properties to arsenious acid, of which, with the metal, it appears to be a mechanical mixture.

ANALYSIS.—When boiled in water, arsenious acid is dissolved, and the appropriate tests may be applied. When a small portion is gently heated in a reduction-tube, a ring of arsenious acid is obtained as well as a ring of metallic arsenic. With soda-flux, a well-defined metallic crust is procured, possessing the characters already described, (ante, p. 274.) This compound is used for destroying flies as well as vermin.

Fly-water is a name applied to solutions of various arsenical compounds in water. One mixture of this kind is formed by dissolving one part of arseniate of soda and two parts of sugar in twenty parts of water. Paper soaked in this solution, and dried, is used for poisoning flies; and, perhaps, this is the safest form in which arsenic can be used for such a purpose. The bodies of these insects thus poisoned become saturated with arsenic. Dr. Galtier states that about one drachm of the flies yielded a number of deposits by the employment of Marsh's process. (Toxicologie, i. 374.)

A case of poisoning by fly-water, in which two grains and a half of arsenious acid destroyed the life of an adult in thirty-six hours, has been lately reported in the Medical Gazette, (vol. xxxix, 116.)

ARSENIC ACID.

This is an artificial product almost entirely confined to the chemical laboratory. Orfila states that it is a more powerful poison than arsenious acid, but he does not adduce any cases in support of this opinion. Dr. Christison does not mention it; and I have not been able to find any case of poisoning by it in the human subject. Dr. Glover ascertained that four grains of this acid, dissolved in two drachms of water and introduced into the stomach of a stout rabbit, killed the animal in four hours, with the symptoms of irritant poisoning, and an affection of the nervous system. (Ed. Med. and Surg. J. lviii. 121.)

Treatment.—The hydrated oxide or acetate of iron would be more likely to act as an antidote in poisoning by arsenic acid—owing to the greater solubility of this compound, and its tendency to combine with the oxide.

Analysis.—Arsenic acid is a white uncrystalline deliquescent solid. 1. It is not volatilized on platina foil, by the flame of a lamp. 2. It is very soluble in water, forming a highly acid solution. 3. It is precipitated of a dull red colour by nitrate or the ammonio-nitrate of silver. In all of these characters it differs from arsenious acid. 4. It yields readily an arsenical sublimate with charcoal. 5. It yields abundant deposits with copper and muriatic acid, or in Marsh's apparatus. It is precipitated, although slowly and of a pale yellow colour, by sulphuretted hydrogen gas. In these properties it resembles arsenious acid.

ARSENIATE OF POTASH.

The arseniates of potash and soda must be regarded as active poisons, although there are but few instances on record in which life has been destroyed by them. Dr. Christison states that, in the course of his reading, he has met with only two reported cases of poisoning by arseniate of potash. (Op. cit. 284.) M. Bouley administered this salt to seven horses, from the effects of which they all died. On inspection, it was observed that there was well-marked inflammation of the stomach, intestines, and bladder, and there were ecchymoses in the left ventricle of the heart. The contents of the viscera in one horse, yielded no traces of the arseniate—a fact probably to be ascribed to the violent diarrhœa from which the animal had suffered. (Orfila, Toxicologie, i. 452.) An attempt at murder by the arseniate of potash was the subject of a trial in France in 1844. This poisonous salt had been maliciously put into a bottle of wine. The prosecutor swallowed a mouthful, and, from finding the liquid very bitter, she spat out the greater portion. His wife also tasted it, but drank only a very small quantity. In the course of the night, the prosecutor was seized with severe colic, vomiting, general prostration of strength and somnolency. The wife suffered from similar symptoms. The medical man who had been called to them, finding that but a small quantity of wine had been taken from the bottle, referred the symptoms to indigestion. The next morning the prosecutor gave a portion of the suspected wine to a dog: the animal suffered from violent vomiting and convulsions, and died in four hours. The wine was then analysed by M. Chevallier, and found to contain about one drachm of arseniate of potash to a pint. A person, in whose possession a large quantity of arseniate of potash was found, was tried for this nefarious attempt to poison, but he was acquitted; his counsel, M. Chaix d'Est-Ange, contending that it was impossible that the prosecutor and his wife could ever have *tasted* the wine, for, had they swallowed any portion of it, they would have been infallibly poisoned! This very strong assertion is reported to have been made on the authority of MM. Devergie, Barse, and Payen. (Journal de Chimie Médicale, 1845, 524.) Admitting the analysis of M. Chevallier to be correct, one ounce of the wine would not have contained more than four grains of the salt, corresponding to about *two grains of arsenic acid.* Admitting that even so much as an ounce was swallowed by the two persons, there are no medical facts to justify the assertion that two grains of arsenic must always of necessity destroy the lives of two adults. Here it will be perceived that, because the parties did not die from this dose of the poison, it was argued in the prisoner's favour, not that he was innocent of poisoning the wine, but that the prosecutor and his wife could not have swallowed it! To this erroneous medical assumption, contradicted as it is by numerous cases where individuals have recovered after having swallowed considerably larger doses of arsenic (ante, p. 269,) the prisoner appears to have owed his acquittal!

TREATMENT.—A solution of an arseniate is copiously precipitated by the hydrated oxide, or a solution of acetate of iron: hence these substances might be administered with a fair prospect of benefit.

ANALYSIS.—Arseniate of potash is a white deliquescent substance, fixed when heated, and very soluble in water. The same tests may be applied to it as to ARSENIC ACID. In order to separate the whole of the arsenic, the liquid may be acidulated with muriatic acid, and a current of sulphuretted hydrogen gas passed into it.

The BINARSENIATE OF POTASH is known under the name of *Macquer's neutral arsenical salt;* and the liquid known as *Pearson's* solution, which is still used medicinally in France, is a mixture of one grain of arseniate of soda to one ounce of distilled water. The alkaline arseniates, mixed with sugar and spread on paper, are used as fly-poisons, and may thus occasion accidents.

SULPHURETS OF ARSENIC.

There are several kinds met with in commerce—ORPIMENT or YELLOW ARSENIC, and REALGAR or RED ARSENIC. They are very poisonous in consequence of their containing a large proportion of free arsenious acid; this sometimes amounts to as much as from 30 to 70 per cent. of their weight. They are not often used as poisons. Orpiment has, however, given occasion to several criminal trials in England.

Orpiment and realgar are employed in the arts, and are procurable by artisans with the most destructive facility. On one occasion, a quantity of red powder, brought to me by a mechanic as iron-rust, which he was carrying loosely in his waistcoat pocket, turned out to be realgar! From the brilliant colours of these compounds, they were used in painting, dying, paper-staining, and even in the colouring of toys and sweetmeats for young children! It is remarkable that, under these circumstances, accidents are not more frequent.

Arsenic is used in some of the yellow dyes (orpiment;) and at a trial a few years since, in which only a minute portion of this poison had been detected in the liver, it was objected that the arsenic might have been imbibed from a dyed handkerchief in which the medical witness had wrapped the viscus! The colour of the handkerchief did not, however, happen to suit this ingenious hypothesis. Paper is sometimes dyed green with arsenite of copper, and yellow with orpiment. In these cases arsenic may be detected as in the process for solid organic substances, (ante, p. 302.) On the continent, these tinted papers have been largely used as wrappers for snuff and tobacco; and it has been seriously debated, whether poison might not be thus imparted to these extensively-used substances. This is very improbable, as the arsenic in the paper is in an insoluble form. It may be proper to mention, that the yellow dye of sulphuret of arsenic is immediately discharged (rendered white) by ammonia or caustic alkali. Some dyes containing lead are similarly affected; but the yellow dye of arsenic undergoes no change when touched with a solution of hydrosulphuret of ammonia, while that of lead is instantly turned of a deep brown-black colour. (See Ann. d'Hyg. 1843, p. 358.)

The sulphurets of arsenic are commonly said to be *insoluble* in water; but, according to Guibourt, 150 parts of boiling water will dissolve one part of the yellow sulphuret, and one part and a half of the red sulphuret.

It is in the state of yellow sulphuret that arsenic is so commonly found in the stomach after death when the body has been buried for a long period. This arises from the action of sulphuretted hydrogen, generated by decomposition, on the white arsenic taken during life. In some instances, the coats of the stomach and intestines may present deep yellow patches from this change taking place in the tissues. This conversion of white to yellow arsenic, is, in general, only partial. In a recent instance I found it complete: but the body had been buried for nearly two years, and the liquid in the stomach contained a large quantity of hydrosulphuret of ammonia. Some remarks have been elsewhere made on this change (ante, p. 296,) which gave rise to an important question, in the case of *Reg.* v. *Johnson*, (Liverpool Lent Assizes, 1847.) It was stated in evidence, that yellow spots were found on the coats of the stomach in cases of poisoning by arsenic: and the learned judge charged the jury against the assumption of poisoning, on the ground, that within *two or three days* after death no yellow spots had been found in the stomach of the deceased. The earliest period at which I have known this conversion to occur, was twenty-eight days after death. I have kept white arsenic, spread as a paste upon a putrefying stomach, under a bell-glass, for ten days, in summer, without observing this change of colour. Any cause which leads to the production of sulphuretted hydrogen in the stomach, may, however, give rise to the change at a much earlier period after death; but it is by no means usual to find it, as a result of putrefaction, within less than a week. In a recent case, in which a small dose of arsenic caused death, the mucous coat, only *twenty-one hours* afterwards, had a yellow colour, which was ascribed to the conversion of the arsenic to sulphuret. (Med. Gaz. xxxix. 116.) The fact that this colour is not apparent on inspection, cannot, however, be regarded, under any circumstances, as negativing the presumption of death from arsenic. M. Decourdemanche states, that when the sulphurets remain long in contact with organic matters, they become changed into arsenious acid and sulphuretted hydrogen. (Galtier, i. 378.) I am not aware of any facts in support of this view: it is certainly not borne out by what is discovered in the dead body.

SYMPTOMS AND APPEARANCES.—The sulphurets of arsenic produce *symptoms* and *appearances* after death similar to those caused by arsenious acid; but the dose required to destroy life must vary according to the proportion of arsenious acid with which the sulphuret happens to be mixed. This is not a common form of poisoning; the intense colour of the poison would lead to suspicion. It was with orpiment that Mrs. Smith was poisoned at Bristol in 1835. (Med. Quart. Rev. July 1835, p. 390.) Its colour might cause it to be mistaken for mustard.

Orpiment has been known to cause death by *external* application as a depilatory, (see Annales d'Hygiène, 1834, 459;) a result which might be expected from the quantity of arsenious acid with which it is mixed. There is a form of depilatory used, which consists of one part of orpiment, twelve parts of quicklime, and ten parts of starch, made into a soft paste with water, (Pereira, i. 218,) the use of which must always be attended with danger.

TREATMENT.—Hydrated sesquioxide of iron has been employed as an antidote in poisoning by the arsenical sulphurets; but it is not easy to perceive how, at a temperature of 98°, any chemical action of an antidotal kind can be exerted between these bodies. The promotion of vomiting with the exhibition of mucilaginous liquids can alone be trusted to.

ANALYSIS.—The powdered sulphurets yields a solution of arsenious acid on boiling them in water acidulated with muriatic acid. They readily give the well-known sublimates of metallic arsenic, both with soda-flux, silver, and in the hydrogen apparatus. They also yield a deposit of arsenic when boiled with copper and muriatic acid. Orpiment is insoluble in muriatic acid, but it is readily dissolved by caustic potash. *Organic mixtures.* The sulphuret being insoluble in water, it is in general easily separated mechanically by allowing the matters mixed with it, to become dry upon bibulous paper. If the sulphuret cannot be separated mechanically, the organic matter suspected to contain it, should be dried and boiled with nitro-muriatic acid to dryness. Any sulphuret will be found, as arsenic acid, soluble in water. Another impure sulphuret, sold as *King's yellow*, is composed, according to Dr. Christison, of sulphuret of arsenic, lime and sulphur. It is highly poisonous, and is extensively sold as a pigment. A case of alleged poisoning by this substance has been already related, (ante, p. 146.) The patient died of bronchitis. The sulphuret of arsenic is easily separated from it by digestion in caustic alkali.

IODIDE AND BROMIDE OF ARSENIC.

Of these preparations it is unnecessary to say more than that they are very active poisons, but nothing is known concerning their operation as such on man.

ARSENURETTED HYDROGEN.

This is a gaseous poison of arsenic, producing, when respired in small quantity, very serious effects upon the system. It has already occasioned death in at least three instances. The gas is an artificial product, and is formed in a chemical laboratory in various ways,—one method has already been described in speaking of Marsh's process; and its highly poisonous properties render it necessary that caution should be used in the employment of this mode of testing. The gas is most effectually decomposed, and prevented from diffusing itself, by passing it into a solution of nitrate of silver. (See ante, p. 264.) This form of gaseous arsenical poisoning has been hitherto purely accidental. Gehlen, a German chemist, was killed by accidentally breathing a small quantity. Suspecting that the gas was escaping from some part of the apparatus he was using, he applied his nose for the purpose of detecting it; and although he respired but a very small quantity, probably a few hundredths of a grain only (Brande) he was seized in about an hour afterwards with vomiting, shivering, and great prostration of strength. He died on the ninth day. The most complete history of this kind of poisoning has been published by Dr. O'Reilly, of Dublin. He has been kind enough to forward me the particulars of one case.

A gentleman, for the sake of experiment, wished to respire about one hundred and fifty cubic inches of hydrogen gas. It unfortunately happened that the sulphuric acid, which he used for making the hydrogen, was largely contaminated with arsenic; and immediately after respiring the gas, he was seized with giddiness and fainting, constant vomiting of a greenish-coloured matter, and dull pain in the epigastrium. There was also complete suppression of urine. He died in about six days. On dissection, the liver and kidneys were found of a deep indigo colour,—the mucous membrane of the stomach was easily separated; and there were two distinct patches of inflammation in the greater curvature. There was a quantity of reddish-coloured fluid effused in the chest, and it is an interesting fact, that Dr. O'Reilly, on examining about ten ounces of this fluid, was enabled by the use of Marsh's process, to detect arsenic in it. From experiments made on the sulphuric acid, it is supposed that the deceased must have inhaled a quantity of arsenic equivalent to about twelve grains of arsenious acid.

A third case of poisoning by this gas occurred in England, in December 1836. A young gentleman was killed by respiring the gas, evolved from a mixture of arsenic, zinc and sulphuric acid. Death did not take place until twenty-four days after the accident. It appears that in this instance but a very small portion could have entered into the lungs.

The detection of arsenic in the serous liquid of the chest, proves that this poison is eliminated not merely by the natural secretions, but also in morbid effusions. Dr. Chatin has recently proposed this as an additional means of diagnosis in poisoning by arsenic. A blister was applied to the chest of a female labouring under the effects of this poison. About ten drachms of the serum were collected, and this quantity contained sufficient arsenic to give sixteen well-marked metallic deposits by the use of Marsh's apparatus. (Journ. de Chimie, 1847, 329.)

Analysis.—The chemical properties of this gas have been already described. (See Marsh's Process, ante, p. 281.) It is colourless, possessed of a disagreeable odour resembling that of garlick, inflammable, burning with a bluish-white flame, and an abundance of white solid vapour. In burning, it is converted to water and arsenious acid. On cold surfaces it deposits metallic arsenic, hydruret of arsenic, arsenious acid, and water. (For the characters of the deposits, see page 283.) It is decomposed by chlorine, forming muriatic acid and chloride of arsenic: also by those metallic solutions the metals of which have a weak affinity for oxygen. Paper soaked in a solution of nitrate of silver and held over the gas, is immediately blackened. If it be passed into the solution, the silver is reduced and arsenious acid is dissolved. The gas is not soluble in water, and the air of water decomposes it. At a red heat the metal is entirely deposited, and hydrogen escapes. This is applied as an adjunct test in Marsh's Process. It is known from most other gases in being totally absorbed by a solution of sulphate of copper (Gregory.) On the assumption that this gas is formed of three equivalents of hydrogen and two equivalents of arsenic, and that its specific gravity is 2·695, it contains by weight 96·2 per cent. of arsenic; and as 100 cubic inches would weigh 82·17 grains, every cubic inch will contain more than 8-10ths of a grain of arsenic in a finely-divided state! It is therefore one of the most formidable poisons with which we are acquainted. No treatment can save life when it has been once respired.

CHAPTER XXV.

ACTION OF MERCURY IN VAPOUR—OF LIQUID MERCURY—CORROSIVE SUBLIMATE—TASTE AND SOLUBILITY—SYMPTOMS—ITS EFFECTS COMPARED WITH THOSE OF ARSENIC. SLOW OR CHRONIC POISONING—SALIVATION FROM SMALL DOSES OF MERCURIAL MEDICINES—FROM OTHER CAUSES, AGGRAVATED BY DISEASE—CASES—CANCRUM ORIS—DIAGNOSIS—EFFECTS OF EXTERNAL APPLICATION—POST-MORTEM APPEARANCES—ANOMALOUS CASES—QUANTITY REQUIRED TO DESTROY LIFE—PERIOD AT WHICH DEATH TAKES PLACE—FATAL DOSE—TREATMENT.

General Remarks.—METALLIC MERCURY, in the state of *vapour*, is well known to be pernicious to health, producing paralysis, tremors of the limbs with emaciation, and other disorders of the system; but this is a form of poisoning which does not require medico-legal investigation. As it has been elsewhere stated (ante, p. 20,) *liquid mercury* appears to be entirely destitute of poisonous properties. Numerous cases are recorded in which this substance has been swallowed with impunity. One was the subject of a report to the Westminster Medical Society in November 1842. The individual in this case laboured under obstinate constipation which ended fatally. Five days before death, half a pound of liquid mercury had been swallowed as a remedial agent; no ill effects followed. On an inspection of the body, the mercury had wholly disappeared. Much larger quantities of the metal have been given without injurious consequences. In a case of obstinate constipation, after trying without effect all the common cathartics, Dr. Borgstedt prescribed for a female, æt. 42, two pounds of mercury, to be swallowed at intervals. The metal remained nine days in the body, and was perceptible to the feel through the abdominal parietes. The last portions of metal were passed by stool on the fourteenth day. Only five-sixths of the quantity administered were recovered. Slight salivation appeared about this time, but this after-effect was speedily subdued. (Casper's Wochenschrift, April 12, 1845, 249.) In the same journal, Dr. Kerstein relates a somewhat similar case, in which, under an attack of ileus, he gave to a man, æt. 28, two pounds of quicksilver, in four doses, six ounces at each dose. Croton oil was then prescribed, and after eight days the bowels were moved, the greater part of the metal being passed unchanged, if we except some portion which had been converted into black oxide. (30 Mai, 1846, 355.) Many similar cases might be quoted, all tending to show that metallic mercury is inert.

The most important mercurial poison is CORROSIVE SUBLIMATE; but there are some other preparations of the metal, as calomel, the nitric oxide, and the acid nitrates of mercury, which have occasioned death.

CORROSIVE SUBLIMATE.

This substance is usually known under the chemical name of BICHLORIDE OF MERCURY; but, according to some distinguished authorities, it is a chloride. To prevent any confusion from scientific chemical nomenclature, the old and popular name of corrosive sublimate is here used. This compound is not often taken as a poison. In the coroner's report for 1837–8, there were about fifteen cases of mercurial poisoning, in twelve of which corrosive sublimate was the poison taken. It is commonly seen under the form of very heavy crystalline masses, or of a white powder. It is freely retailed to the public at the rate of twopence for from one to two drachms; if exceeding this quantity the price is sixpence per ounce. This may guide the witness when he has to judge of the quantity taken, by the price paid.

Taste and Solubility.—The *taste* of corrosive sublimate is powerfully austere and metallic, so that no poisonous quantity of it can be easily swallowed without the individual becoming immediately aware of it. It is very *soluble* in water, hot or cold, and speedily sinks in it, in which properties it differs strikingly from arsenic. I have found by experiment that one hundred grains of a cold saturated solution hold dissolved at a maximum, ten grains of corrosive sublimate; and it is stated by most chemists that two parts of boiling water (212°) will dissolve one part of the poison. It is also readily dissolved by alcohol and ether; the last body takes up one-third of its weight, and has the property of abstracting it from its aqueous solution,—a principle which is sometimes advantageously resorted to for separating the poison when dissolved in organic liquids. It is soluble without change in nitric and muriatic acids, and it is a fact of some medico-legal importance, that common salt renders it much more soluble in water.

[There is a discrepancy of opinion as to its solubility; Thenard says, it is soluble in 20 parts of temperate water, whilst Orfila says that it dissolved in 11. Devergie states that three parts boiling water will take up one of the salt. Dr. Davy found it soluble in water at 57°, in the proportion of 1 in 5.4. When it is finely powdered and thrown in water, a part falls to the bottom, and part floats. When in an impalpable state, almost the whole will float, and it requires much shaking and stirring to precipitate it.—G.]

SYMPTOMS.

The symptoms produced by corrosive sublimate, generally come on immediately or within a few minutes after the poison has been swallowed. In the first place, there is perceived a strong metallic taste in the mouth, often described as a coppery taste, and there is during the act of swallowing, a sense of constriction almost amounting to suffocation, and burning heat in the throat, extending downwards to the stomach. In a few minutes violent pain is felt in the abdomen, especially in the region of the stomach, which is increased by pressure. Pain in the abdomen is, however, sometimes wholly absent. There is nausea, with frequent vomiting of long stringy masses of white mucus, mixed with blood; and this is accompanied by profuse diarrhœa. The countenance is sometimes swollen and flushed, in other cases it has been pale and anxious. The pulse is small, frequent, and irregular, becoming scarcely perceptible as the symptoms become aggravated. The tongue is white and shrivelled,—the skin is cold and clammy, the respiration difficult; and death is commonly preceded by syncope, convulsions, or general insensibility. The internal parts of the mouth, with the lips are swollen, and often present the appearance as if the cavity had been washed with a solution of nitrate of silver. Suppression of urine has been frequently noticed among the symptoms. It existed in a well-marked case of poisoning by this substance at Guy's Hospital. The patient lived four days, but did not pass any urine during the whole of this time (p. 321, post.) This symptom was observed in an interesting case reported by Dr. Wegeler (Casper's Wochenschrift, Jan. 10, 1846, p. 30,) in which a youth, ætat. 17, swallowed three drachms of the poison, and died on the sixth day. During the last three days, no urine was secreted. The case was otherwise remarkable from the fact that no pain was experienced on pressure of the abdomen, and that the pulse underwent no change until shortly before death. In another case reported by Dr. Herapath, in which a scruple of corrosive sublimate in solution was swallowed, suppression of urine and ptyalism came on on the third day, and the patient died on the ninth day, (Lancet, Dec. 13 and 27, 1845, pp. 650, 698.) In a case observed by Mr. Morris, the quantity of urine secreted was small, and it produced a scalding pain when voided. (Prov. Med. Jour. Nov. 18, 1843, 126.) In this instance there was no purging. The symptoms are much modified when the poison is given in small doses at certain intervals for some days or weeks. There are, after each dose, colicky pains, with nausea and vomiting. There is general uneasiness and depression; the salivary glands become inflamed and painful; the tongue and gums are red and swollen, sometimes ulcerated, and there is fœtor of the breath. A deep blue line, like that observed in poisoning by lead, is sometimes found around the edges of the gums. The patient experiences difficulty of swallowing and breathing. The constitutional effects are indicated by cardialgia, diarrhœa, dyspnœa, hæmoptysis, cough, general tremor of the limbs, and paralysis, with slow fever and marasmus, under which the patient sinks.

Its effects compared with those of arsenic.—This poison differs from arsenic: 1, in having a well-marked taste; 2, in producing violent symptoms within a few minutes; and 3, in the fact of the evacuations being more frequently mixed with blood. The symptoms produced by corrosive sublimate, in the first instance, resemble those of cholera; if the individual should survive several days, they are more like those of dysentery,—tenesmus and mucous discharges mixed with blood being very frequently observed.

Salivation.—One of the most marked effects of slow or chronic poisoning by mercurial preparations is salivation or ptyalism: indicated by swelling of the salivary glands and an increased flow of saliva. This is by no means a necessary symptom in cases of acute poisoning by corrosive sublimate, although it not unfrequently shows itself about the second or third day. In some instances, the patient dies too rapidly for this effect to follow. In a case related by Dr. Venables, in which two drachms of the poison had been taken, and the woman survived eight days, this symptom did not exist. (See also a case, p. 323, post.) In another, reported by Mr. Wood, (Ed. Med. Surg. Jour. li. 141,) in which half a tea-spoonful of the poison was taken, salivation was profuse in the course of a few hours. In a case which occurred at Guy's Hospital, in February 1843, where two drachms had been taken, salivation commenced in four hours (p. 322, post:) but this is by no means the earliest period. Dr. Percy relates an interesting case of poisoning by corrosive sublimate, in which the saliva was flowing profusely an hour and a half after the woman had taken a dose of thirty grains. (See Med. Gaz. 1843, i. 942.) In these early cases, it is alleged that fœtor of the breath is absent, but most practitioners will look chiefly to the production of salivation as a symptom. The local action of the poison is, in some instances, sufficient to account for the abundant flow of saliva independently of the influence of the absorbed mercury on the salivary organs. In Mr. Morris's case, in which half a drachm of the poison in powder was placed by the female on her tongue, the saliva flowed abundantly from the mouth, and the lips were much swollen. (Prov. Med. Jour. Nov. 18, 1843, p. 127.) This was undoubtedly due to the local irritant action of the poison.

In the *chronic* form of poisoning, when the dose has been small and frequently repeated, we may generally expect to meet with salivation accompanied by fœtor of the breath and sponginess and ulceration of the gums. Should the person survive some time, this symptom is more commonly met with than not; but in looking for it as an indication of mercurial poisoning, a medical jurist must remember, that some persons are wholly unsusceptible of this condition. On the other hand, there are cases in which the salivary glands are most easily excited, so that the usual innocent doses of mercurial medicines have been known to produce salivation to such a degree, as to cause death. Facts of this kind are of some importance, since charges of malapraxis may be easily raised in respect to them. Dr. Christison mentions a case in which two grains of calomel destroyed life by the severe salivation induced, as well as by ulceration of the throat. Another was mentioned to me by a pupil, in 1839, in which five grains of calomel killed an adult by producing fatal salivation. From some cases related by Mr. Samuel of Newark, it appears that two grains of calomel divided into three powders, were given in the proportion of one powder daily, (two-thirds of a grain,) to a little boy aged eight. This small dose produced the most violent salivation, sloughing and exfoliation, from which he was some weeks in recovering. In another instance, a little girl aged five, took daily for three days, three grains of mercury and chalk powder. Her mouth was severely affected, sloughing ensued, and she died in eight days (see page 318.) In a third case, three grains of blue pill given twice a day for three days, making eighteen grains, were ordered for a girl aged nineteen, who complained of a slight pain in her abdomen. Severe salivation supervened, the teeth separated, and she died in twelve days. With respect to the effects of corrosive sublimate, Dr. Christison states that three grains of this substance in three doses, caused violent salivation. (Op. cit. 408.) When this state results from the use of mild mercurial medicines in small doses, the severe effects may be referred to idiosyncrasy (ante, p. 37.) A person may die under these circumstances:—either from simple exhaustion or from extensive sloughing of the fauces with exfoliation of the bones. When an individual has recovered from the first effects of acute poisoning by corrosive sublimate, he may die at almost any period from these secondary consequences.

It is generally admitted by toxicologists, that salivation may be intermittent, *i. e.* that it may cease and reappear without more mercurial poison, or any mercurial preparation, being given in the interim, although such cases are rare. As a matter of medical jurisprudence, this important question was brought to an issue at the trial of *Butterfield*, at Croydon, in 1775. The deceased was supposed to have been killed by the administration of small doses of corrosive sublimate; and the fact of his having become salivated at or about the time of the alleged administration, was regarded as a proof of poisoning. In the defence, it was urged that the deceased had been salivated two months previously, under a common mercurial course,—and although the salivation had ceased for that period, it was probable that this was nothing more than a recurrence of the former:—it did not prove that there had been any fresh administration of mercury in the interim. There was a difference of opinion on this point among the witnesses, as there probably would be in the present day, if each relied upon his own individual experience. However, one of the witnesses had known salivation to recur without a fresh exhibition of mercury after the long interval of *three months*, and the prisoner was acquitted. Cases are reported of salivation recurring after intervals even longer than this. One is quoted by Mr. Swan, in which salivation recurred after an interval of six months. (On the Action of Mercury, p. 4. 1847.)

[Although evidence is in favour of these intermittent salivations, still some doubts exist on the subject. Dr. Christison observes "granting the ptyalism to be in every instance mercurial, it would require much better evidence than any practitioner could procure, to determine the fact, that mercury had not been given during the supposed interval."—G.]

This symptom is not necessarily connected with the exhibition of mercury, and therefore, when taken alone, it can never furnish evidence of mercurial poisoning. It may come on spontaneously from disease in the salivary organs; or it may arise from a simple mechanical cause. Dr. Mulock has communicated a case to the Dublin Hospital Gazette, in which profuse salivation was occasioned by the introduction of a set of artificial teeth. (Sept. 15, 1845, p. 35.) It may also be produced by many other substances besides the preparations of mercury. Thus it has been known to follow the use of the preparations of gold, copper, bismuth, lead, antimony, iodine, iodide of potassium, croton oil, opium, prussic acid, sulphuric acid, arsenic, colchicum, foxglove, and cantharides. Some have asserted that fœtor of the breath, a brassy taste in the mouth, and spongy and ulcerated gums, would, indicate the salivation caused by mercury: but these characters have been equally met with in the salivation produced by arsenic and bismuth. (Prov. Med. Journ., Oct. 22, 1845, p. 638.) A case in which the question of diagnosis from fœtor was material, has been reported by Mr. Harding. (See Lancet, June 13, 1846, p. 654.) A very important investigation, involving the evidence of poisoning by mercury, as derived from salivation and fœtor of the breath, took place at Nottingham, in February 1845, in the case of a girl named *Wilmot*. Although she suffered from symptoms of mercurial poisoning, the verdict returned was, that she had died from disease of the brain. It is important to state that, according to the researches of pathologists, salivation is not so readily induced by mercurial preparations in young subjects as in the adult. Dr. Beck says that, in his experience, only one instance has occurred in which a child, two years old, was salivated by five grains of calomel given in three doses in twelve hours: in two days the usual symptoms appeared. The child had been labouring under hooping-cough for several weeks, and was a good deal reduced. For other instances, see page 312, ante; but notwithstanding these cases, it is true as a general principle that young subjects are salivated with great difficulty, and at the same time the effects of mercury upon them, are frequently more energetic and uncertain than they are in the adult. Hence mercurial preparations should be always administered with great caution, when the strength of a child has been reduced by disease. In this state of constitutional depression, a single cathartic dose of calomel may sometimes prove fatal. (Dubl. Med. Press, May 12, 1847, p. 296; also Amer. Journ. Med. Sciences, April 1847, p. 509.)

Profuse salivation from mercury dependent on morbid causes.—In addition to the facts already detailed, respecting death from excessive salivation under the use of small doses of calomel, there are certain morbid conditions of the body which appear to have the effect of increasing the action of this medicine on the salivary glands. This kind of acquired idiosyncrasy exists especially in that form of disease called granular degeneration of the kidney, which is characterized in its early stage by albuminuria. On this subject Dr. Craigie says, the great objection to the employment of any preparation of quicksilver for the cure of renal dropsy, consists in the fact, that the use of the mineral is known to render the urine albuminous, to increase the inflammatory state of the system, and to induce the disease, the effects of which it is expected to remove. Another evil is, that in persons labouring under symptoms of granular kidney, a very small quantity of mercury induces ptyalism, and renders the mouth tender and most painful. (Practice of Physic, ii. 1148.) This he considers to depend much on the fact, that patients of this description have in general, if not always, been subjected previously to the full influence of the mineral in repeated courses. For these reasons, in his view, mercury should never be exhibited without the previous full trial of other remedies, as even assiduous watching will not always succeed in preventing bad effects. Dr. Christison informs me he has repeatedly observed that mercurial action (salivation) is in these cases brought on by unusually small doses of the compounds of mercury, or unusually soon; and the action, in these cases, has been very violent, although not uncontrollable. Mr. Harrison, of Reading, has communicated to me two cases in which the serious effects of mercury under similar circumstances were well-marked. A woman labouring under granular disease of the kidney, rubbed into her side about one drachm of strong mercurial ointment: profuse salivation, with exfoliation of the jaw, followed. The other case occurred to Dr. Cowan. He gave two grains of mercury with chalk, to a woman labouring under atrophy of the kidneys: the most intense salivation was produced. Other instances of the injurious effects of the preparations of mercury in this disease, might be easily brought forward: the opinion based on these observations is, that, as a general rule, the use of mercurial medicines is, in these cases, improper. The question, however, is by no means settled; for some eminent practitioners look upon the fear of injurious consequences, in combining mercury with other diuretics in such cases, as unfounded. Dr. Gregory states that opinions vary as to the propriety of employing mercury: instances of recovery are recorded after severe salivation, but the general impression is, that the mercurial influence is prejudicial rather than salutary. (Practice of Medicine, 692.) From these facts it is obvious that a practitioner may, under these circumstances, find himself charged with malapraxis; and the following case, in which an unlicensed practitioner was implicated, may prove in this respect instructive. It was referred to me in December 1845, by the magistrates of Reading. A man, labouring under disease of the kidneys, had placed himself in the hands of a person, who promised to cure him. Part of the treatment consisted in the administration of small doses of calomel. Profuse salivation came on, and the patient, not finding himself relieved, applied to a regular medical practitioner. In about a fortnight afterwards he died, and a coroner's inquest was held, in order to determine whether his death had not arisen from improper treatmenton the part of the person whom he consulted. Some pills and powders supplied to the deceased by the inculpated party, were brought to me for chemical examination. The powders consisted of drachm-doses of bicarbonate of soda. The active ingredient in the pills was calomel, each pill containing not more than three-quarters of a grain.

The following evidence came out at the adjourned inquest:—The deceased, J. W., who had been employed at the gas-works, became indisposed in the early part of the month of November, and sought the advice of a surgeon. He continued under his care about a fortnight. Not finding himself relieved by the medical treatment he had undergone, he went, on the 7th of November, to a Mr. Lamb, an unlicensed practitioner, who, after interrogating him as to the nature of the complaint, gave him the powders and pills. Directions were given in writing by Mr. Lamb to the deceased to take *half a pill three times a day;* but his wife, being unable to read, administered the medicine in double doses, and in a few days salivation was produced. He was five days under the care of Mr. Lamb, and the 12th of November an order was given for the attendance of the surgeon of one of the districts of the Union. This gentleman visited him until the time of his death, the 25th of November.

Admitting that the ingredients were well mixed, the quantity of calomel prescribed would be equal to *three-eighths* of a grain at each dose; making, therefore, a grain and an eighth per diem. It appeared that the deceased began to take the pills on Friday, the 7th of November, and continued to take them until he was placed under the care of the Union surgeon on the 12th; *i. e.* five days. Had the written order been followed, he would thus have taken rather less than *six grains* of calomel during this period; but, in consequence of a mistake, he took *eleven grains and a quarter*, i. e. *two grains and a quarter daily* for five days. In favour of the prisoner it was elicited, that when he was informed of profuse salivation having arisen from the use of the pills, he ordered the deceased to discontinue them; but the order does not appear to have been obeyed. On an inspection of the body, the gums were found ulcerated, and the mucous membrane of the tongue, mouth and fauces, was in a state of intense irritation. The left kidney was found much enlarged, double its natural size; externally smooth, of a deep purple colour: on making a section, it was much firmer than natural; the cortical and tubular portions distinct; the cortical mottled, the tubular of a dark red colour, and much indurated; but little blood escaped on its division. The right kidney was smaller, and the morbid changes in it were less distinctly marked; ureters normal. After hearing the evidence of several medical witnesses, the jury returned a verdict that deceased died from natural causes.

There are several points in which this case is of great interest to the medical jurist. In the first place, it became a question as to the existence of an acquired idiosyncrasy to the influence of mercury, under a diseased condition of the kidneys. I do not know that any attention has been paid to this subject by toxicologists. I have not been able to find any reference to it in the toxicological work of Orfila. Evidence was given at the inquest to show that the views of the profession were not uniform respecting the use of mercury in this disease; and the opinion of Dr. Williams, in favour of the practice, was quoted by the solicitor for the prisoner. The coroner left this to the jury as a still unsettled question; since it could not be said that any practitioner, who chooses to employ calomel (provided he keeps within proper limits as to the dose,) is to be precluded from its use. This would do away with freedom of judgment in the practice of medicine, and be placing medical practitioners in the dangerous position of having to answer charges of manslaughter for every untoward result in their practice. Medical experience is proverbially uncertain; and we daily find that persons recover from diseases or accidents under diametrically opposite modes of treatment, each practitioner regarding the other as acting upon most dangerous and improper principles.

The fact may, therefore, be clearly admitted, that, as a general rule, mercurial preparations, even in small doses, are liable to produce excessive salivation in persons affected with renal disease; but this would not bind every medical man to avoid their employment; nor, in the event of an untoward result, would it justify a charge of manslaughter against him; because, in fact, his individual experience may not have led him to concur in the correctness of the general opinion. I am here considering this question in the abstract, and as it may affect hereafter any medical practitioner; for principles once correctly laid down, will apply to all persons who practice medicine, whether with or without a diploma. The great question, as it affected the prisoner, in this case, was not so much whether the use of calomel was or was not proper under the circumstances, as whether it was likely to have been the cause of excessive salivation and death, in the *doses* in which he ordered it to be taken. It is clear, that if a medical man prescribe a certain dose of a powerful medicine (as prussic acid,) or of a medicine likely to act powerfully on the body, as mercury in diseased kidneys, written directions being given so that there may be no room for mistake,—and double the quantity be administered, it would not be just to make him responsible for the result. If the rule were otherwise, what practitioner would be safe for a single day from the mistakes made by patients? Therefore, the question in reference to the prisoner was, not so much whether death had really been caused by the quantity of mercury actually given; but whether the excessive salivation and death would have been likely to arise from the doses which he actually prescribed.

The medical evidence rendered it apparent that salivation had followed in the usual period after the exhibition of mercury; that it was profuse, and had the effect of exhausting the patient; that for thirteen days before death (on the 20th) the salivary discharge had diminished, but the mouth was still very sore; and on the 23d, two days before death, the gums were still ulcerated, and bleeding at intervals. These facts tended to show that the exhaustion produced by the salivation might have accelerated death, although the proximate cause was the disease under which the deceased was labouring.

It appears that eleven grains and a quarter of calomel were taken, during a period of five days, instead of the quantity ordered, *five grains and a half.* Admitting the existence of an idiosyncrasy to the effects of mercury in persons affected with renal disease, the quantity taken would fully account for the occurrence of violent salivation; although it would be only a moderate dose under other circumstances: and perhaps even half the quantity (*i. e.* the doses prescribed) might have produced serious effects, although it is impossible to affirm that it would have necessarily occasioned death. To prescribe a grain of calomel daily, under the circumstances, for five days, could hardly be pronounced an act of gross ignorance and criminal carelessness; yet, without the expression of a positive opinion of this kind, the party prescribing the medicine, could not be committed on a charge of manslaughter. The verdict of the jury was probably base on many circumstances favourable to the prisoner, The quantity of calomel prescribed by him, was small. Admitting that it was not an advisable medicine in cases of renal disease, evidence was adduced to show that some difference of opinion among medical men might fairly exist on the subject. He had given written directions respecting the doses, but these, through the inattention of the deceased's wife, had been doubled. The patient had exposed himself to night-air while taking the medicines; and when informed of the effects of the pills, the prisoner had ordered them to be discontinued. These facts necessarily weighed strongly in his favour, and led to his discharge. The case, however, is of importance, in showing the injurious effects which may occasionally arise from the use of even small doses of mercury in disease of the kidneys.

It may be proper to state that, in albuminuria, the kidneys appear after death larger than natural, of a dark or chocolate colour, and they are evidently gorged with blood. During life the disease is known, among other symptoms, by the coagulability of the urine. In granular degeneration the surface of the kidney is generally mottled or speckled: in the earlier periods of the disease the organ may be found larger than natural, and of softer consistence, while in the advanced stages it may be contracted and hard. The outer or cortical portion is commonly disorganized; it is granular, and of a pale yellow colour. (See Gregory, Practice of Medicine, 691.)

Cancrum oris.—Corrosive sublimate, as well as other mercurial preparations, is liable to produce *gangrene of the mouth* and fauces, a state which may equally occur from spontaneous causes: death is commonly the result. In a case of this kind, supposing any mercurial preparation to have been given medicinally, it may become a serious question whether death actually resulted from the mercury acting as a poison, or from natural disease. Several fatal cases have occurred within the last few years, among young children; and the subject has become a matter of inquiry before coroners. Although salivation and its sequelæ are not common among young children, as an effect of mercurial preparations, yet it is clear, from the cases already cited (p. 312,) that small doses of mercury may have a most violent effect upon them, and render the suspicion of poisoning probable. Of two children, whose deaths became the subject of investigation under these circumstances, one was affected with hooping-cough, and the other with measles. Powders containing calomel, were prescribed in both cases,—gangrene of the mouth supervened, and the children died. There was some reason to believe, from the evidence, that the mercury had really produced the effect attributed to it, at least in one of the cases. It is proper to remark, that this kind of disease, gangrene of the mouth, has been observed to occur in children, to whom no calomel, nor any mercurial preparation whatever, had been exhibited:—the subjects have been chiefly young infants, badly fed and clothed, and generally labouring under, or recovering from, fever, small-pox, measles or hooping-cough. It is, however, far more common as a sequela of measles than of other exanthemata, and it is always connected with a depressed state of the vital powers. Many cases of this kind are reported by Dr. Hennis Green (see Lancet, Dec. 1839.) The disease is commonly described under the name of "*Cancrum oris.*" A case occurred in August, 1840, in which a charge was made against a medical practitioner of having caused the death of a child, aged four years, by administering an overdose of some mercurial preparation. The child was labouring under hooping-cough, and some medicine was prescribed; on the fourth day, the child complained of soreness of the mouth, the teeth became loose and fell out, the tongue and cheek were very much swollen, and the child died in the course of a few days from gangrene in the left cheek. The answer to the charge was, that not a particle of mercury had been exhibited,—a fact clearly proved by the production of the prescription-book of the medical attendant. This, then, was an instance in which the gangrene proceeded from spontaneous causes; and yet it is almost certain, that had any mercury been proved to exist in the medicine prescribed, a verdict affecting the character of the practitioner would have been returned! In several instances, where but a small quantity of mercury had been exhibited, the disease and death were referred to it;—the cause appeared so obvious, that the general impression could not be shaken by the medical statement, that similar cases had occurred where no mercury was used. Sometimes the case may really be of a doubtful nature. A boy aged three years, while suffering under an attack of measles, took small doses of mercury by the prescription of a physician. Soon after the administration of the medicine, the child became worse, the mouth became inflamed, dark and discoloured, and the teeth dropped out. He died in a few days. A practitioner who had been called in, pronounced that the child had been excessively salivated. Mercury had undoubtedly been taken, and it was proved, that the person who had dispensed the medicine, did not weigh it. An inquest was held, and a verdict returned, that the child had died from an overdose of mercury. Mr. P. H. Holland, of Manchester, has forwarded to me the report of an interesting case of cancrum oris. The child, aged six years, had taken four grains of calomel in two doses with rhubarb. Swelling and sloughing of the salivary glands came on, and the child died in ten days. The disease was not caused by the mercurial. Mr. Holland heard at the same time, of six cases of excessive salivation in young children from common doses of calomel, almost indicating an influence of an epidemic character. (See also an interesting case by Mr. Dunn, Med. Gaz. xxxiii. 57, and Br. and For. Med. Rev., October, 1844, p. 542.) It is worthy of remark, that in cases of this description, the popular opinion is generally supported by that of some medical practitioner, showing how easily members of the profession, as well as the public, are led to refer the effects to what in many instances is only an *apparent* cause. An important case of this kind, in which the medical witness relied upon the "mercurial fœtor" as characteristic and distinctive, will be found in the Lancet (June 13, 1846, p. 654.) The following is a doubtful instance: a child aged about four years, suffering from hooping-cough, took, according to a prescription obtained from a dispensary, three grains of calomel on the 29th of October: this dose was repeated five times between that date and the 7th of November following. About this time the right cheek became much swollen, and there was great difficulty in opening the mouth, with very offensive breath. The gums and inside of the cheek became ulcerated, and on the 16th a sphacelus appeared on the right cheek of the size of a shilling, which rapidly extended, and the child died on the 28th. This was considered to be a case of cancrum oris from spontaneous causes; but it would be difficult to say that the calomel had not here been the exciting cause. At any rate, it cannot be denied that there was reasonable ground for suspicion.

Diagnosis.—Are there any means of distinguishing gangrene as a result of disease, from the gangrene produced by mercury? A diagnosis has been founded on the allegation, that when the gangrene is caused by mercury it is uniformly diffused over the gums, tongue, and internal parts of the cheek; while, when of a spontaneous character, it is restricted to one patch or spot, beginning on the inside of the cheek as a hard swelling. This, however, is a very weak criterion. In a case recorded by Dr. Christison, in which it resulted from mercury, the gangrene was observed to occur on the skin, near the mouth on each side: it thence spread over the whole of the cheek, and destroyed life in eight days. In general, however, it begins in the mouth or in the throat, and spreads onwards. Besides, it is quite possible that the spontaneous gangrene may present a diffused character. There is no certain diagnosis; at least, there are no general rules to guide a medical opinion: each case must be judged of by itself. The time of the occurrence of the symptoms, after taking the medicine, may be sometimes a good criterion; but this is not always applicable,—for, by mere coincidence, the symptoms may supervene without being connected with the medicine. Then again, the symptoms may not have been caused, but only aggravated, by the continued use of mercurials. The fact of the dose of calomel, or other preparation, having been small, is not of itself an obstacle to the admission of the view, that it has really caused the gangrene; since cases have been already related, which show that in certain constitutions, small doses of mercury have produced the most alarming and unexpected effects. Unless, then, a medical witness is prepared to assert, that no such idosyncrasy could have existed in the case under investigation, it will be considered, when other facts concur, that the smallness of the dose is no answer to the charge of the medicine having produced these serious consequences. It is also not improbable, that the diseases under which such subjects have been observed to labour, may aggravate the effects of mercury so administered, and render them more prone to this affection.

Effects of external application.—Cases of poisoning by the *external* application of corrosive sublimate are rare; it acts energetically through the unbroken skin, producing severe local and constitutional symptoms, and even death. Two fatal cases of this kind have been reported by Mr. Ward of Bodmin. (Med. Gaz. iii. 666.) A man aged twenty-four, rubbed over every part of his body, one ounce of corrosive sublimate, mixed with six ounces of hog's lard, for the purpose of curing the itch. In an hour, he experienced excruciating pain in the abdomen and over the whole of his body;—he said he felt roasted alive,—he also suffered from intolerable thirst. The skin was found completely vesicated. He died on the eleventh day, having laboured under bloody vomiting, purging, and tenesmus. Ptyalism did not show itself until thirty-six hours after the application of the poison. The brother of the deceased, aged nineteen, rubbed in the same quantity of the poison. The symptoms were much the same, but more aggravated. There was constant vomiting, with complete suppression of the urine, and frequently bloody stools;—the ptyalism was not so severe. He died on the fifth day. On inspection the stomach was found much inflamed, and partially ulcerated. The small intestines were also greatly inflamed throughout; and the lower portion of the colon and rectum were in a state of mortification. The bladder was contracted, and without urine. Thirty large worms were found alive in the stomach and intestines! (For another case, see Niemann Taschenb. der Arzneiw. 452.) Death from the external application of corrosive sublimate has been the subject of a trial In this case there were the usual symptoms of irritation, and the stomach and intestines were much inflamed, (*Reg.* v. *Welsh*, Worcester Summer Assizes, 1845; Med. Gaz. xxxvi. 608.) The readiness with which this poison acts through the skin is proved by the following circumstance. M. Cloquet plunged his hands into a concentrated solution of corrosive sublimate, in order to remove some anatomical preparations.

He did not wash his hands afterwards; and in about eight hours he was attacked with severe pain in the abdomen, constriction in the chest, painful respiration, thirst, nausea, and ineffectual attempts at vomiting. Under the use of diluents these symptoms were removed, but for eight days he suffered from pain in the epigastrium. (Galtier, 1-567.) This case should serve as a caution to anatomists.

Mr. Annan has reported a singular instance of the local action of corrosive sublimate, which appears to have led to death after a very long period. In Jan. 1845, a man aged 38, a shepherd had been employed several hours daily in washing sheep, which were affected with cutaneous disease, with a solution of two drachms of corrosive sublimate in twenty ounces of water, in which muriate of ammonia was also dissolved. He was suddenly seized with sickness, vomiting, constitutional irritation, and after the lapse of five days with ptyalism, although not severe. He did not recover for a fortnight. In six weeks he experienced a similar attack from the same cause, and this left behind it great debility and emaciation. He resumed his occupation, but was attacked with wandering pains in the joints and diseases of the bones,—as if from the secondary effects of mercury; and he died fourteen months after the first attack. (Med. Times, July 25, 1846, p. 331.) Of ten of the sheep two died shortly after the application.

Salivation is a common effect of the external application of this poison. Dr. Guerard has seen ptyalism produced as a result of three corrosive sublimate baths, one ounce of the poison to about ten gallons of water taken at intervals of three days: but the effects produced by the solution, are never so powerful or so dangerous as those which arise from the application of the poison in the form of ointment. There are many ointments sold by quacks for the treatment of skin diseases, which contain corrosive sublimate.

When any mercurial preparations are used as escharotics, ptyalism may very speedily follow. Breschet observed this effect in twenty-four hours from the application of the acid nitrate of mercury, to the cervix uteri.

POST-MORTEM APPEARANCES.

These, as in the case of arsenic, are chiefly confined to the alimentary canal. Corrosive sublimate, however, affects both the mouth and fauces; the mucous membrane is softened, of a white or blueish grey colour, and sometimes inflamed; that lining the œsophagus is similarly affected, and partially corroded and softened. The mucous membrane of the stomach is more or less inflamed, sometimes in patches; and there are masses of black extravasated blood found beneath it. Occasionally the whole cavity has a slate-grey colour from the partial decomposition of the poison by the membrane itself; beneath this the mucous coat may be found reddened. This grey tint of the mucous membrane has been considered by some to be indicative of the action of the poison on the living mucous membrane; but it is not always present. A case occurred at Guy's Hospital, in which the mucous membrane was simply inflamed, and very much resembled the condition presented in cases of arsenical poisoning. The coats of the stomach are sometimes corroded, and so much softened that they cannot be removed from the body without laceration. Similar appearances have been met with in the intestines, especially in the cæcum. In a case by Dr. Herapath, in which a scruple was taken, and death occurred on the ninth day, the mucous membrane of the stomach was softened, but there were no well-marked appearances of the action of the poison in this organ. The cæcum had been the seat of the most violent inflammation, the whole surface being of a deep black-red colour, and there were patches of sloughing in the coats. (Lancet, Dec. 27, 1845, p. 700.) Perforation of the stomach is very rare as an effect of this poison: there is, I believe, only one case on record. Certain morbid changes have been met with in the urinary and circulating organs; and Mr. Swan states that he has found the ganglia and branches of the sympathetic, inflamed, but these changes are not by any means characteristic of this variety of poisoning. Appearances in the alimentary canal, like those just described, have been seen, not only where the case has terminated fatally in a few hours, but where it has been protracted for six, eight, and even eleven days. (Chaussier, Recueil de Mémoires, 363.)

I here subjoin a few cases which have fallen under my notice, as they may serve to illustrate better than any summary, the varieties in the symptoms and post-mortem appearances occasionally met with in poisoning by corrosive sublimate.

Cases.—J. W., aged 38, a stout healthy-looking man, was admitted into Guy's Hospital on the 10th of February, 1843. It was ascertained, that about 10 o'clock on the morning of that day he put into his mouth about two drachms of corrosive sublimate, in coarse lumps, which he masticated and swallowed: he then drank about a pint of water. A surgeon, who saw him soon afterwards, administered to him four eggs. He then vomited, and a piece of corrosive sublimate, about the size of half a nut, was found in the vessel; but this, it was supposed, had not been swallowed. When admitted into the hospital, the following symptoms were observed:—there was great depression of the system; the extremities were quite cold; the respiration natural; the pulse scarcely perceptible; the tongue swollen, as well as the lips. The man was sensible; and complained of constriction of the œsophagus. The albumen of several eggs was immediately administered to him. At 2 P.M. the lips were excessively swollen and tender, as well as the gums; there was incipient salivation, with pain down the œsophagus, into the stomach. The act of swallowing gave rise to much pain. The man had vomited several times, and brought up a quantity of a yellow-coloured matter, interspersed with blood: he complained of slight pain in the abdomen. There were spasms of the lower extremities; the knees were drawn up; the pulse was small and scarcely perceptible; the tongue was white, and so much swollen that he could not protrude it from his mouth; the skin was rather warmer. During the day, the man swallowed about two pints of milk, and the albumen of twenty-four eggs.

Feb. 11.—He was attacked with several violent fits of hiccough during the night; there was great pain upon pressure over the region of the stomach. There was much greater difficulty of swallowing, and violent pain in the head. The lower lip had become considerably swollen, and a small blister had formed on the inside of it. The pulse was small and wiry, scarcely perceptible. He had vomited several times; the bowels were fully relieved, and the motions were of a green colour; his feet were very cold, and there was general yellowness of the skin.

12th.—The skin was still very yellow; the pulse was sharp, and with difficulty felt; the swelling of the lips had subsided. The patient had vomited some green matter; his bowels were well opened, and the fæces were of a dark colour mixed with blood. There was great pain in the region of the stomach, with difficulty of swallowing. The extremities were warm.

13th.—The power of swallowing had slightly returned; but the man still complained of a burning sensation in the œsophagus: the pulse continued the same. No urine had been passed since his admission. He had had very little sleep during the night; the hiccough continued. The pupils were much contracted; the extremities cold; the bowels had been opened several times: motions were streaked with blood, and once or twice they consisted entirely of blood, mixed with mucus. There was not much pain in the abdomen, but the sickness still continued. The lips had almost returned to their natural size; the tongue was swollen, so that it could not be protruded, and it was covered with a white fur. At 4 P. M. symptoms of delirium supervened; the pulse was small, and scarcely perceptible. The delirium increased, and he was obliged to be forcibly confined to his bed.

14th.—At 6 A. M. there was stertorous breathing at intervals, and the man was evidently becoming much worse. His lower eyelid dropped as well as his jaw; and towards noon he seemed to have great constriction of the œsophagus. Slight consciousness still remained: there was no power to speak; but he moved his arm about, as if aware of what was going forward. His bowels had not been relieved since six o'clock the preceding evening; no urine had been passed. The stertorous breathing continued until shortly before three o'clock, P. M. when he died.

The body was examined twenty-two hours after death. There was general rigidity, but no appearance of decomposition; the atmosphere was cold and dry. The peritoneum was healthy, and contained about one ounce of a straw-coloured liquid. At the greater curvature of the stomach, within four inches of the pylorus, there was a large patch of inflammation, about the size of the palm of the hand. The mucous membrane was highly injected with red blood, and presented marks of inflammation throughout. The slate-grey colour, which is sometimes seen in cases of poisoning by corrosive sublimate, did not here exist. There was no appearance of corrosion or ulceration in any part. The duodenum and jejunum were healthy: there was slight inflammation of the mucous membrane about the lower two-thirds of the ileum; and this was more marked towards the termination of the intestine. Near to the cæcum there were several patches of inflammation. The whole of the large intestines were highly inflamed; and there were several small spots of ulceration, about the size of a pea. The liver was enlarged and congested: the gall-bladder contracted, and containing scarcely any trace of bile. The spleen was of its natural size, congested and firm: the peritoneal covering was thin and closely adherent to its substance. The pancreas was healthy; the kidneys of a natural size and consistence, rather florid; the cortical portion presented minute red points, more especially between the infundibula, probably the results of commencing inflammation. The right kidney had a cyst on its posterior surface, about the size of a marble, containing a clear fluid. The bladder was much contracted; the mucous membrane slightly injected; it contained about half an ounce of turbid urine.

The mucous membrane of the œsophagus was reddened from inflammation, but had no other abnormal appearance. In the chest there were found old adhesions of the pleura; the substance of the lungs was œdematous, somewhat firm and doughy; at the base of the right lung there was inflammatory œdema. The bronchial membrane was inflamed throughout its whole extent; and within the tubes, there was an abundance of frothy mucus. The bronchial glands were slightly enlarged. The pericardium contained about six drachms of a straw-coloured fluid, part of which was reserved for a chemical examination. The heart was rather smaller than natural, but the cavities and the parietes were healthy: some partially-coagulated blood was found in the organ. The head was not examined.

A chemical analysis was made of the contents of the stomach, of the blood, spleen, and serous liquids found in the pericardium and peritoneum, but no mercury was detected.

In comparing this with other cases of acute poisoning by corrosive sublimate, the following points deserve notice:—1st, With regard to *symptoms*. Some of these—as, for example, constriction of the œsophagus—were such as are commonly met with in cases of poisoning by corrosive sublimate: in others, there were some peculiarities. Thus salivation began to make its appearance in about *four hours* after the poison was swallowed, which, I believe is an unusually early period for this symptom to be observed. One of the most strongly-marked symptoms was, however, the complete *suppression of urine.* This has been occasionally witnessed before; but here the suppression continued for upwards of three days, *i. e.* during the whole of the period that the patient survived. The effect of the poison on the nervous system was clearly indicated by the stertor and other symptoms which immediately preceded death. With respect to *post-mortem appearances*, it is remarkable, that although life was so long protracted, there were no marks of corrosion, ulceration, or any tendency to perforation of the stomach. This organ was simply inflamed, and very much resembled the condition which it presents in cases of arsenical poisoning. The slate-grey colour of the mucous membrane was here entirely wanting. It is remarkable that the duodenum and jejunum should have entirely escaped the action of the poison, and that the chief effect on the small intestines was seen on the lower part of the ileum. The other appearances do not require any special notice.

The exact quantity of poison which the deceased took, could not be correctly ascertained; there was reason to suppose, however, that he could not have swallowed less than *two drachms.* This quantity is fully sufficient to destroy life; although persons have been known to recover from the effects of much larger doses. It will be seen that notwithstanding the active treatment speedily adopted, and the free exhibition of the antidote—albumen,—the fatal progress of the symptoms could not be arrested. The entire absence of the poison from the stomach showed that the treatment had been very effectual in removing it.

The case here reported suggests an important subject of reflection in relation to the evidence of poisoning, when the fact of poison having been taken, requires to be established by medical evidence. Chemical proofs of the nature of the poison were here entirely wanting, so far as related to the inspection of the body: the post-mortem appearances were not very characteristic; and yet there could be no doubt that this was really an instance of acute mineral poisoning. Had this been a case involving a question of murder, it would have required but a very slight exercise of ingenuity for a person to have drawn from the absence of poison in the body, a plausible argument that it was not a case of poisoning, but one of natural disease! The actual occurrence of well-marked cases of this description should, therefore, convey a practical caution to a medical jurist; and not lead him to assume too hastily that the presence of poison in the stomach is the only *certain* evidence of death from poisoning, or to admit, because it is not detected, that the symptoms and post-mortem appearances might be safely referred to disease. (G. H. Rep. April 1844, 24.)

Another case of poisoning by corrosive sublimate occurred in the hospital during the summer of 1846. S. C——, aged 25, a woman of intemperate habits, was admitted labouring under symptoms of poisoning by corrosive sublimate, which she had taken dissolved in spirits of camphor. It appears that about five o'clock in the morning, she put into her mouth a table-spoonful of a mixture containing corrosive sublimate dissolved in spirits of camphor; but her husband seized her by the throat and prevented her from swallowing but a very small portion of it; she spat the greater part out again. She immediately felt a strong coppery taste, with burning heat in the mouth and throat, attended with difficulty in swallowing. In about five or ten minutes she vomited. When admitted into the hospital she appeared much excited. There was no collapse or coldness of the extremities: on the contrary, they were warm and moist. Her face was very much disfigured by the projection of the lips, which had become considerably swollen and excessively tender. On examination, the fauces were not found to be much injured, the action of the poison having been chiefly confined to the mouth. Some of the fluid had run down from each corner of the mouth, producing patches of redness externally: the mucous membrane of the lips and tongue had a whitened aspect; there was a great deal of retching, and she vomited, but not violently. The pupils were dilated, and the conjunctivæ inflamed. There was painful headach; the pulse, which was at first unaffected, subsequently quickened to 112, and became irregular; her breathing was unimpaired. She passed her water freely, in large quantity; urine pale, not bloody. There was no diarrhœa, nor any *pain* at the stomach. There was great thirst, with occasionally a tendency to sleep. The pain in the throat afterwards extended to the chest: the submaxillary glands became enlarged and tender; the gums were unaffected; no salivation nor fœtor. Two dozen eggs and milk were given to her. The matter vomited was but slightly tinged with blood. 10 P. M. Has passed no motion; no pain at the stomach, nor sickness; mouth and throat painful; able to pass urine; pulse full, not so jerking; is very restless. Enema of gruel and castor-oil. 24th. Much better; lips nearly the natural size; mucous membrane peeling off; gums sore; throat painful; sick occasionally; bowels unrelieved by enema. 25th. Bowels opened last night; stool dark-coloured, unhealthy, and offensive; liquid portion like porter, the solid pale, not tinged with blood; feels better; pulse 80; sleeps well; no headach; still complains of her throat; unable to swallow much food; urine passed freely; mucous membrane still peeling off; pain extending further down the chest. After this, the woman gradually improved, but her mouth and gums remained tender for four days.

While the symptoms in this case were those of general irritation, it is worthy of remark that the most prominent effects of the poison were confined to the mouth. It is probable that but a small quantity of the poison had been swallowed: a most violent local action had, however, been exerted by it on the mouth and lips. The lining membrane of this cavity was quite white, but not abraded: the lower lip completely everted, and swollen probably to an inch in thickness. It is remarkable that the gums became sore only after a few days, and that there was no salivation nor any mercurial fœtor in the breath. It will be also observed that in this case there was no diarrhœa nor any suppression of the urinary secretion. Some doubt might have been entertained whether the poison had really reached the stomach, but for the fact that mercury was detected, although in small quantity, in the matter ejected from the stomach, both before and after the exhibition of albumen. A portion of the liquid which the woman had swallowed was examined, and found to be a strong solution of corrosive sublimate in spirit with camphor.

H. H——, a man of dissolute habits, in a fit of despondency, swallowed a quantity of corrosive sublimate under the following circumstances:—On the morning of April 16th, 1846, about eleven o'clock, he called at a druggist's shop and asked for two pennyworth of corrosive sublimate *in solution*. The druggist took some of the powder out of a bottle on the end of a spatula (about one drachm,) and rubbed it down with something fuming, (hydrochloric acid?) and about half a pint of distilled water, and gave it to the man, who soon afterwards swallowed the *greater part* of it. He experienced great heat and soreness of the fauces and œsophagus in the act of swallowing the poison; and was shortly afterwards seized with violent vomiting and purging. For these symptoms the druggist who was applied to, exhibited some liquor calcis, but with what object it is difficult to say, as the resulting compound, red oxide of mercury, is poisonous. The man was brought to the hospital at half-past eleven, and he was then labouring under the following symptoms:—Cold extremities, and a general lividity of the surface, especially about the backs of the hands, cheeks, and forehead, which presented a purplish-black appearance. The surface of the body was warm, and skin soft. The functions of the brain and nervous system were not materially affected. He appeared anxious and desponding, and was affected with rigors and frequent chattering of the teeth, without any sense of cold in any part of the body; pupils at first natural, and obedient to variations of light; they were subsequently contracted; functions of respiration normal at first; pulse about 100, small and feeble; feet and hands cold; violent vomiting and purging, the former occurring at first every two or three minutes, subsequently every half hour. The vomited matter consisted of mucus tinged with blood, and mixed with albumen, which had been administered. Purging every hour throughout the day: the stools, which consisted at first of little else than mucus and blood, became more and more fæculent. The tongue was covered with a greyish-white crust, resembling the effect of nitrate of silver; its margin was of a bluish colour. There was great heat with soreness of the mouth, fauces, and œsophagus, especially during the act of swallowing any liquid; the gums had receded from the base of the crowns of the teeth, and presented a leaden-blue margin; there was some pain at the scrobiculus cordis; the abdomen was, however, generally free from pain; there were occasional cramps of the lower extremities. On the 18th, the symptoms were ameliorated; stools more fæculent; vomiting and purging abated. The vomited matters were of a blackish-green colour; pulse 90, soft and small; scarcely any sleep. On the 19th, vomiting and purging at intervals of three quarters of an hour; he became delirious. On the 20th, the symptoms were aggravated; the blue line round the border of the gums was still observed; there was general tremor, with cramps of the extremities. He died at nine o'clock P. M., on the 20th, having survived the effects of the poison rather more than four days. It is worthy of remark, that there were no symptoms of salivation, nor was there any secretion of urine during the whole progress of the case. The body was inspected sixteen hours after death. The membranes of the brain were rather opaque; and there was a considerable quantity of fluid beneath the arachnoid. The whole brain was remarkably pale. Lungs appeared somewhat consolidated, as if from early pneumonia. Heart healthy; its cavities filled with colourless firm fibrin. Kidneys: secreting structure congested; mucous membrane of one pelvis had a spot of ecchymosis. Bladder empty and contracted; some small pink spots on its mucous membrane. Œsophagus: the mucous membrane had a vermilion hue. Stomach presented a pink colour on its inferior surface, near its middle. Duodenum tolerably healthy. Small intestines healthy, lined with thick yellow mucus. Cæcum and ileo-cæcal valve showed signs of the most intense inflammation; some portions of a deep purplish-black colour, with patches of sloughing mucous membrane, tinged green by fæces. Colon and rectum also exhibited traces of the most violent inflammation, especially the ascending and transverse colon. Here were found oval patches of sloughing mucous membrane, about the size of small almonds, and tinged green by fæces passing over them.

This case is somewhat similar to that reported at page 321. In both there were the ordinary signs of irritation, and in both there was complete suppression of urine, continuing throughout the whole duration of the case. The former case terminated fatally in nearly four days: in the present instance the duration was four and a half days. There was, however, one remarkable difference in the symptoms, which shows what anomalies may be encountered in the action of this poison. In the first case salivation made its appearance so early as *four hours* after two drachms of the poison had been taken: in the present case one drachm was taken; and although the individual survived nearly *five days*, there was no salivation at any time. In the first case, the large intestines were highly inflamed throughout: in the present instance the inflammation was most marked about the cæcum and ileo-cæcal valve.

QUANTITY REQUIRED TO DESTROY LIFE.

This is a question which it is somewhat difficult to answer with any degree of certainty, since it is only by accident that the quantity taken can be ascertained, and the fatal effects must vary according to many circumstances. A child, aged three years, died in twenty-three days from the effects of twelve grains of corrosive sublimate. The *smallest* dose which is reported to have destroyed life was *three* grains. This was also in the case of a child, and the quantity was accurately determined from the fact of its having been made up by mistake for three grains of calomel, which the physician intended to order. (This case is referred to in the Lancet, 1845, p. 297.) A very loose and imperfect report either of the same or of a similar case is given in the Ann. d'Hyg., 1835, i. 225. It is stated that three children lost their lives. It is probable that, under favourable circumstances, from three to five grains, or even less, would destroy an adult. In the case of *Reg.* v. *Robertshaw* (Carlisle Lent Assizes, 1845,) there is reason to believe that two or not more than three grains were taken, and proved fatal to an adult. (Med. Gaz. xxxv. 778.) In its power as a poison, it is therefore somewhat similar to arsenic. Persons have, however, been known to recover who have taken very large doses, when remedies were timely administered, or vomiting was promoted. In an instance reported in the Journal de Pharmacie, a man recovered in three days after having taken one drachm of the poison. In the Medical Gazette (xiv. 63,) Dr. Booth mentions a case where *an ounce* of corrosive sublimate has been swallowed after a full meal; and by timely vomiting the subject of this rash act escaped with comparative impunity. In a case by Dr. Percy (Med. Gaz. xxxi. 942,) a girl, aged seventeen, mixed thirty grains in coarse powder with water in a tea-cup, and then swallowed the liquid. A considerable quantity remained in the cup. Symptoms of poisoning came on, but the girl recovered. The reporter doubted whether any of the poison reached the stomach. A most interesting case of recovery after *forty grains* had been taken in whiskey, under circumstances favourable to its fatal operation, *i. e.* on an empty stomach, is recorded by Dr. Andrews. (Cormack's Journal, Feb. 1845, 102.) The patient was a woman of sixty-five. The actually smallest dose required to destroy an adult, under ordinary circumstances, cannot therefore be determined at present from any reported facts. Judging from the effects produced by small quantities used medicinally, possibly the average fatal dose may not differ widely from that of arsenic, *i. e.* two or three grains. Experiments on animals cannot settle this question,—observations on man must be purely accidental, and the cases that have yet occurred are not conclusive on the point. The doses either go beyond, or fall short of, what may be fairly assigned as a possibly fatal dose. That one person has actually recovered after swallowing *forty grains* of this poison, does not show that twenty or thirty grains might be taken with impunity; although this absurd argument was used in respect to prussic acid by some of the medical defenders of Tawell! If true of one it would be equally true of all poisons. In order to determine the minimum fatal dose of a poison, its known general effects, either medicinal or otherwise, must be taken into account, or a medical opinion will assuredly lead to error.

PERIOD AT WHICH DEATH TAKES PLACE.

In an acute case, an individual commonly dies in from one to five days. But death may take place much sooner or much later than this. A person has been known to die from the effects of this poison in eleven hours (Christison, 402;) and in one instance of a child two years old, by whom twelve grains had been taken, death probably occurred in six hours. (Niemann's Taschenbuch, 451.) A case is reported in which a child, aged seven, was killed in three hours by eighteen grains of corrosive sublimate. In the following instance the period, although inferential, was probably even shorter. It is reported by Mr. Illingworth. (Med. Gaz. xxxi. 557.)

A man, æt. 30, was found dead on the 4th December, 1842, at half-past seven A. M. He had vomited some half-digested food, mixed with blood and mucus. On a shelf near him was a drinking horn, containing about three drachms of corrosive sublimate. It was ascertained at the inquest that he had died from the effects of this poison. He had put water into the drinking-vessel, and had probably swallowed the poison while thus loosely suspended,—the exact quantity taken could not be ascertained. The deceased was last seen alive at half-past eleven the preceding evening; therefore only eight hours before he was found dead. When discovered the face and the extremities were cold. From all the circumstances it was inferred that, even admitting the deceased to have taken the poison immediately after he was last seen alive, he could not have been dead for less than six hours. This would carry the duration of the case to *two hours* from the time of taking the poison.

The following, communicated to me by Mr. John Welch, is remarkable as being, I believe, the most rapidly fatal instance of poisoning by corrosive sublimate on record. On Friday evening, June 19, 1846, about five minutes after six, Mr. Welch was summoned to attend a man who was said to have taken poison, but of what kind was not known except that its appearance was white. A period of less than five minutes had elapsed before Mr. Welch saw the patient. On his arrival he found the man totally insensible; the breathing somewhat laborious (almost inclined to stertor;) pulseless at the wrist, with a countenance pallid, but neither anxious nor distorted. In less than a minute after his arrival, without any struggling or convulsions, he died. On examining the mouth, lips (interiorly,) and fauces, they presented an entirely uniform appearance in colour, " such as would immediately follow the application of a strong solution of lunar caustic;" in other words, they were white and corroded. This led to a suspicion of the nature of the poison. Near the body, was found some portion of the contents of the stomach, of a watery character, tinged with mucus. This was collected: it amounted to about a large teaspoonful, showing how little he had vomited. The room, as well as his person, were closely examined, but no trace, either of poison, or of paper which might have contained it, could be found. The fire, which was almost out, had a piece of twisted paper on it; but this was so burnt to a cinder, that it fell to pieces in trying to remove it. The external jugular vein was opened by Mr. Welch when he was first called, as the compression on the brain appeared to be the most urgent symptom: but little blood flowed from the incision. On inquiry, Mr. Welch ascertained the following particulars:—The deceased was a man of intemperate habits, aged about thirty; and on the afternoon of this occurrence had been drinking, which may account in a great measure for the stertor and want of sensibility. Between five and six o'clock he sent his wife out for some porter, and while she was gone he mixed a white powder (corrosive sublimate,) in a milk-cup, with some tea, and, after pouring it into a cup, drank it off. On her return, seeing the froth issuing from his mouth, she taxed him with having taken poison, which he admitted. She immediately raised an alarm, which brought to her assistance some neighbours, who went for medical aid. They were unsuccessful until they found Mr. Welch, and he reached the spot in less than five minutes: so that from the time the first alarm was given, which was at *ten minutes to six* (so sworn to by one of the witnesses,) and the arrival of Mr. Welch, which he noticed to be at *ten* *minutes after six*, not more than twenty minutes could have elapsed; and adding to that another additional five minutes—that being the time his wife was away fetching the porter, which was at a public house only three or four doors off—it appears certain that the death of this man took place in less than *half an hour.* According to the account given by a bystander, the only symptoms of which the deceased complained, were a burning heat in the mouth and great nausea. The ordinary train of symptoms peculiar to this poison do not appear to have presented themselves. Mr. Welch subsequently analyzed, not only the liquid which was in the cup, but also the vomited matters; and the results, in both cases, clearly proved that corrosive sublimate had been taken. An inquest was held, and the jury returned a verdict, "That the deceased had died from the effects of corrosive sublimate, taken whilst in a fit of temporary insanity brought on by repeated intemperance." No post-mortem examination was made, as, from the satisfactory evidence of the cause of death, the coroner did not regard it as necessary. Considering the striking peculiarity of this case, namely, its rapidly fatal termination, this is much to be regretted, as it might have added some interesting facts to our pathological knowledge of the early effects produced by this poison on the body.

Mr. Bigsley has published a case which proved fatal in two hours and a half; but the poison was a solution of the nitrate of mercury, not corrosive sublimate. On the other hand, the case may be protracted for several days: the following summary will not only show this, but will also prove that the time at which the poison destroys life, cannot be inferred from the quantity taken. In a case related by Dr. Venables, two drachms of the poison killed a woman in eight days,—and in another that occurred to Mr. Watson of Edinburgh, about the same quantity destroyed life in six days. In a third, reported by Sobernheim, three drachms did not kill for *eleven* days; while in an instance, referred to by Niemann, (Tasch. d. Arzneiw. 452,) where one ounce of the poison was swallowed, the person did not die until the sixth day. In death from chronic poisoning, the case may be protracted almost indefinitely.

TREATMENT.

If vomiting do not already exist, it must be promoted by the exhibition of emetics. Various antidotes have been suggested for this poison; and among these, albumen both of the yolk and white of egg, mixed with water, is perhaps the best fitted to counteract its effects. This remedy appears to have been beneficial even when it was not taken until some time after the poison had been swallowed. (See Chapter on Antidotes, ante, p. 83.) Gluten may also be used:—this may be prepared by washing flour in a muslin bag, under a current of water. Should the case be urgent, the flour may be at once exhibited in the form of a thick paste mixed with water. Gluten may often be obtained in this way, when albumen is not at hand. M. Bouchardat states that Cullerier saved two hundred patients who had taken an overdose of corrosive sublimate, by making them swallow, in twenty-four hours, from seven to eight quarts of milk, with decoction of linseed and warm water. (Gaz. Méd. 9 Jan. 1847.) These antidotal liquids may be serviceably employed for the purpose of favouring the expulsion of the poison by vomiting, on which the safety of the patient essentially depends.

Among the numerous chemical antidotes proposed, M. Mialhe has recommended the hydrated proto-sulphuret of iron; but Orfila has proved that this substance is totally inefficacious when not given until after the elapse of ten or fifteen minutes from the time at which the poison has been swallowed. Toxicologie, i. 720.) The same objection holds to the hydrated persulphuret recommended by M. Bouchardat.

In all cases, the entire expulsion of the poison from the stomach should be looked to by the practitioner; and albumen or gluten may be given at the same time to aid the efforts of vomiting. The use of the stomach-pump is of questionable propriety; since if the parietes of the œsophagus and stomach be much softened and corroded, very slight force in its employment might lead to perforation. In order to check excessive salivation, Mr. Allison has recommended small doses of chlorate of potash. (See Med. Gaz. xxxviii. 953.)

CHAPTER XXVI.

CHEMICAL ANALYSIS IN THE SOLID STATE—REDUCTION-PROCESS—TESTS FOR CORROSIVE SUBLIMATE IN SOLUTION—THEIR DELICACY—OBJECTIONS TO THEIR EMPLOYMENT—IN ORGANIC LIQUIDS—DETECTION OF MERCURY IN THE TISSUES—IN ORGANIC SOLIDS—CALOMEL—ITS EFFECTS—ITS ALLEGED CONVERSION TO CORROSIVE SUBLIMATE—WHITE PRECIPITATE—OXIDES OF MERCURY—GREY POWDER—RED PRECIPITATE—VERMILION—BICYANIDE OF MERCURY—TURBITH MINERAL—NITRATES OF MERCURY—PROPORTION OF MERCURY IN PHARMACEUTICAL PREPARATIONS.

CHEMICAL ANALYSIS.

In the solid state.—We will first suppose that the poison is in the *solid* state, and in the form of a white powder. 1. A small quantity heated on thin platina foil is entirely volatilized at a moderate heat,—(care should be taken in performing this experiment,)—in this property corrosive sublimate resembles arsenic, but differs in all other respects. 2. It is very soluble in water,—if the water be warmed, the powder will be dissolved instantly. 3. A small quantity of the powder dropped into a white saucer, containing a solution of iodide of potassium, is turned of a bright scarlet colour. 4. Dropped into potash in a similar way it is turned of a yellow colour. 5. Into a solution of hydrosulphuret of ammonia, it is turned black. 6. When a few grains are rubbed on a clean surface of copper, with a mixture of one part of muriatic acid, and two parts of water, a bright silvery stain is produced, which is entirely volatilized by heat. If zinc or tin-foil be used instead of copper, the surface acquires a silvery lustre, and the metal becomes remarkably brittle. 7. When mixed with three or four parts of calcined carbonate of soda, and heated in a small tube similar to that employed in the analysis of arsenic, the metal is reduced; and a ring of bright globules of mercury is formed, while common salt remains in the tube. For the success of this experiment, the materials must be quite dry, and the tube at first gently heated; any undecomposed corrosive sublimate that may be sublimed, should be driven higher up, before finally applying a strong heat, so that the ring of mercury may not be obscured by it. This last experiment is conclusive of the nature of the substance; because mercury, being the only liquid metal, is the only metal which sublimes in globules. If the end of the reduction tube, containing the fused chloride of sodium left as a residue in the decomposition, be filed off, reduced to powder and boiled in water with a little nitric acid, a solution is obtained in which, on the addition of nitrate of silver, chlorine may be proved to exist. The analysis is then complete. The properties mentioned under 1, 2, and 5, are possessed in common by other bodies; but the other characters are peculiar to the persalts of mercury: and when the results agree, render it absolutely certain that the powder must be a persalt of that metal. The action of nitrate of silver upon the solution of the residue will prove that the persalt must have been a *chloride*. There are therefore no *objections* to this mode of analysis. The experiment of reduction will answer with any visible quantity of the poison. An excellent reducing agent has been proposed by Dr. Frampton, namely, purverulent metallic silver. Four parts of this should be used to one of corrosive sublimate. (See Med. Gaz. xxxii. 384.)

The *weight* of the metallic sublimate may be determined in the same way as that of arsenic (p. 274, ante,) and it may be preserved in like manner *i. e.* by hermetically sealing the tube.

In solution in water.—It is very soluble in water, (ante, p. 311,) forming a clear solution, which, when concentrated, has a faintly acid reaction and a strong metallic taste. A few drops of the solution may be first gently evaporated on a slip of glass, and then set aside to crystallize. If it be corrosive sublimate, it forms slender opake silky prisms, sometimes of considerable length, and intersecting each other. When a weak solution of iodide of potassium is dropped on them, they acquire a bright scarlet colour, and chloride of potassium is formed. These characters which may be obtained from the minutest crystal and only one drop of solution, prove that the body dissolved in water, is corrosive sublimate: it is thus distinguished from every other mineral poison, and all other substances whatever.

TESTS.

1. *Potash.*—On adding a small quantity of caustic potash to the solution, a reddish-coloured precipitate falls, becoming yellow by the addition of a larger quantity of alkali. This precipitate, when washed, dried, and heated in a reduction tube, yields a well-defined ring of metallic mercury. The filtered liquid will be found, on being tested with nitrate of silver, to contain chloride of potassium, thus proving that the mercury was combined with chlorine—and that the compound was soluble in water. 2. *Protochloride of tin.*—On adding this test in rather large quantity to the solution, a white precipitate at first falls down, (calomel,) becoming speedily of a slate-grey colour, and afterwards almost black. On warming the liquid it soon becomes clear, while a heavy precipitate, in great part formed of pure metallic mercury, falls to the bottom of the vessel. The mercury may be collected by pouring the liquid on a filter, and afterwards warming the filter; or its presence may be easily demonstrated by pouring the water carefully from the precipitate, and forcing down upon this a slip of bibulous paper;—this absorbs the water from the mercury, and the pressure condenses the metal into one or more well-defined globules. 3. *Sulphuretted hydrogen gas.*—This gives at first a precipitate, partly black and partly white, (chlorosulphuret,) becoming entirely black when the current of gas has been allowed to pass in for some time. *Hydrosulphuret of ammonia* gives a similar precipitate in the solution;—thus clearly distinguishing corrosive sublimate from arsenic. The test acts equally in an acid solution of the salt. The precipitated black sulphuret of mercury, dried and heated with carbonate of soda or metallic silver, easily furnishes a ring of pure metallic mercury. 4. *Precipitation by metals.*—If we acidulate the liquid with a few drops of diluted muriatic acid, and introduce a slip of bright copper, or what is better, fine copper-gauze, it is soon coated with metallic mercury, having more or less of a silvery lustre, especially on friction. On heating the copper in a reduction-tube, the mercury may be obtained in well-defined globules. If, instead of copper, we employ finely powdered silver, as recommended by Dr. Frampton, the results are more satisfac-

tory; and a larger quantity of mercury is procured in the sublimate. Pure tin or finely-laminated zinc may be also usefully employed. While the zinc acquires a silvery whiteness, it becomes brittle, and when broken up and heated in a reduction-tube an abundant sublimate of metallic mercury is obtained. 5. *The galvanic test.*—There are various ways in which galvanism may be applied to the detection of mercury in corrosive sublimate. Dr. Wollaston, on one occasion, employed an iron key and a guinea: he placed a drop of the suspected solution on a surface of gold, and touched it and the gold with the key:—the mercury was deposited on the gold in a bright silvery stain. The following is a ready method of producing the metal. Place a few drops of the solution on a clean surface of copper and slightly acidulate it with muriatic acid:—then touch the copper through the solution with a slip of zinc foil. Wherever the copper is touched by the zinc, the mercury is deposited, and on washing the surface with diluted muriatic acid or ammonia a silvery stain is left, which is immediately dissipated by the heat of a spirit-lamp. The experiment may be modified by twisting a slip of zinc round a slip of bright copper, or copper-gauze, and introducing them into the liquid:—any change of colour or tarnish is very apparent on the copper. Mercury is deposited on both metals. A surface of gold with zinc foil is, perhaps, more delicate than a surface of copper as a test of the presence of mercury. Applied in a way to be presently explained, it will detect the metal when nearly every other method fails. Other tests have been proposed; but I omit all notice of them, because the foregoing are, in my opinion, quite sufficient for every practical purpose.

Delicacy of the tests.—It may be proper to mention here the relative action of these tests. The protochloride of tin, added to the 8640th of a grain of corrosive sublimate, dissolved in one drop of water in a minute tube, produced a dark grey discoloration, from which in twenty-four hours a black substance separated. This, however, was in such small quantity that it was impossible to determine whether it did or did not contain any mercury. This test added to one fluid-drachm of water holding dissolved the 144th of a grain of corrosive sublimate, produced great turbidness, and a finely-divided precipitate fell down in twenty-four hours. From this precipitate it was found impossible to procure any portion of metallic mercury. On an equal quantity of the same solution, iodide of potassium had no effect whatever; but the hydro-sulphuret of ammonia gave a dark greenish-black colour without any decided precipitate. Coils of gold and zinc foil, and of copper and zinc foil, which failed to detect the 8640th grain in the smallest possible quantity of water, were now introduced into two quantities of liquid containing each the 144th of a grain in one fluid-drachm, and a drop of diluted muriatic acid was added. There was no apparent change for several hours. After twenty-four hours the copper and gold were coated, the former having a bright silvery lustre. The film of mercury on the copper, was so thin that no globules could be obtained on heating the metal in a reduction tube. From the gold and zinc, however, two well-defined globules of mercury, perceptible only by the aid of a lens, were procured. These are perhaps the limits of the application of the tests for this poison; and it would appear, therefore, that by the galvanic test we may procure clear evidence of the presence of mercury in cases where the protochloride of tin fails to separate it in a distinctly recognizable form. With respect to the silver test of Dr. Frampton, it is very delicate, and perhaps only inferior in action to gold and zinc. I found in my experiments that no mercurial sublimate was obtained, when the quantity of corrosive sublimate was the 144th of a grain in one fluid-drachm of water; and this was after long boiling, and several hours' contact. Dr. Frampton does not appear to have operated on less than a grain of the poison, confining himself merely to the effects of dilution; but he found that the silver test enabled him to detect mercury when the quantity of corrosive sublimate was no more than one grain in eight fluid ounces of water. There is no doubt that this test is sufficiently delicate for all practical purposes. Before we employ it, we should satisfy ourselves that the silver contains no mercury, by heating a portion of it in a reduction-tube.

Objections.—Among the above-mentioned tests, there is only one to which any objection can be offered, namely,—3. Sulphuretted hydrogen gas, or hydrosulphuret of ammonia. Either one or both of these re-agents will give a black or dark brown precipitate with several metals, as for example with the salts of lead, copper, bismuth, silver, nickel, and some of the compounds of tin (p. 131, ante.) The precipitate derived from mercury is, however, distinguished from all the others, by the fact that it easily yields this metal when dried and heated in a tube with carbonate of soda or metallic silver. Again, the objection is at once answered by the fact, that the other tests are conclusive of the presence of a *mercurial* salt: it could only apply when the production of a black precipitate from sulphuretted hydrogen was alone relied on, as evidence of the presence of a mercurial salt. Hitherto it will be seen that the analysis has been chiefly confined to the demonstration of the mercury. In speaking of the potash-test, the means of proving the presence of chlorine were pointed out: but the nitrate of silver may be at once added to a solution of the poison itself. It has been said that this is objectionable, because calomel is also precipitated. Admitting that this is the case, still the test is equally applicable; because there is no other soluble salt of mercury which gives a precipitate with nitrate of silver, insoluble in nitric acid, except corrosive sublimate.

In liquids containing organic matter.—The same process of analysis will apply to the vomited matters and contents of the stomach. Masses of corrosive sublimate may be sometimes locked up in thick viscid mucus; and in such cases, the coarse powder being heavy, it may be sometimes separated by simply agitating the viscid liquid in water, and then decanting it suddenly. This poison is decomposed and precipitated by many organic principles, such as albumen, fibrin, mucous membrane,—also by gluten, tannin, and other vegetable substances. Thus, then, we cannot always expect to find it in a state of solution. We must filter in order to separate the liquid from the solid portion; and our first object will be to determine whether any of the poison is held in solution. If the liquid be clear, the protochloride of tin may be used as a trial-test: if an abundant grey precipitate be formed, the poison is probably present in large quantity. I have found occasionally useful, as a trial-test, the galvanic reduction of a portion of the liquid on copper-foil, by means of zinc and a small quantity of diluted muriatic acid. Supposing that one or both of these tests give strong evidence of the presence of mercury dissolved, we may add to the liquid one-half of its bulk of ether in a bottle, and agitate the mixture. On allowing it to stand some time, the greater part of the ether will rise to the surface, holding dissolved the corrosive sublimate, which was contained in the organic liquid. This may be separated by decantation, by a funnel or pipette, and distilled in a retort:—the corrosive sublimate is thus recovered in a pure state. This process, which was first suggested by Dr. Christison, I have found to answer only where the corrosive sublimate was in moderately large proportion. If none should be thus procured, the following method will detect mercury, even when present in very small quantity; and this plan, of course, applies to all those cases where the trial tests fail to give any satisfactory indications of its presence. Cut a slip of fine gold foil, of about one inch in length and one-eighth of an inch in width; it should be just large enough to enter into a small reduction-tube. We then twist round this, in a spiral form, a narrow slip of finely-laminated zinc; acidulate the suspected liquid with a few drops of diluted muriatic acid, and suspend the gold and zinc by a thread in the midst of it. Several such pieces may be at once suspended in the liquid. According to the quantity of mercury present, the gold will be coated with a grey-coloured deposit, either immediately or in the course of a few hours. If at the end of ten or twelve hours the gold retains its bright yellow colour, there is probably no mercury present, or the quantity is exceedingly minute. Supposing the gold to have lost its colour, owing to its having become completely coated, we should remove it and dip it in ether, and afterwards in distilled water, to wash off any corrosive sublimate and organic matter adhering to it: it should then be dried in air without being allowed to touch any surface, and introduced into a reduction-tube. The zinc may be in part dissolved; but as mercury is also deposited on this metal, whereby it is commonly rendered quite brittle, it may be introduced with the gold into the tube. On applying heat, a fine sublimate will soon appear in the cool part of the tube, which, if not perceptible to the eye, may be easily seen, by the aid of a common lens, to consist of minute globules of mercury. Slips of fine copper or copper-gauze, with or without zinc attached, or slips of pure laminated zinc alone, may also be employed as a substitute for gold; but in this case, after removing the metals, it is necessary not only to wash in water, but in a solution of weak ammonia, in order to remove from the mercury any adhering salt of copper. The action of copper is aided by applying heat to the solution. Devergie has recommended tin;—but common tin is said to be often contaminated with mercury. There is no doubt that the silver tests of Dr. Frampton is preferable to the use of tin. According to the experiments of this gentleman, mercury is easily separated by means of silver on long boiling, when the poison is in an extreme state of dilution. (Med. Gaz. Oct. 1843.) I found that a few drops of an organic liquid, containing corrosive sublimate, which was prepared five years before, yielded, on boiling it with metallic silver and muriatic acid, satisfactory evidence of mercury in the course of a few minutes. Dr. Frampton recommends that the deposit should be boiled in potash to remove organic matter, and then digested in ammonia, to dissolve the chloride of silver formed:—but there is an objection to this in the fact, that potash, on boiling, reduces the chloride to oxide of silver, and this, with ammonia, might produce a fulminating compound. The ammonia should therefore be first employed. Simple washing in water will commonly be found sufficient. The reduction by silver would be convenient, when the quantity of organic liquid was small; but otherwise we should prefer the use of gold and zinc. Should there be any difficulty in procuring stout gold-foil for this experiment, copper covered with a layer of gold will answer equally well. Copper foil may be gilt by the ordinary process of electro-gilding, using the cyanide of potassium as the solvent for gold; or slips of copper and fine copper wire or gauze coiled up may be covered with a thin layer of gold, by boiling them for twenty minutes in a solution of chloride of gold, to which crystals of bicarbonate of potash have been added in excess. This mixture must be boiled for half an hour before it is used for gilding; and the copper should be thoroughly cleaned by plunging it into diluted nitric acid, before it is introduced. Platina may be substituted for gold in this experiment, and gold or platina wire may be used instead of the laminated metals; but the great advantage of gold is, that we can note the degree and rapidity of deposition by the loss of its bright yellow colour.

Dilution interferes with the action of the galvanic test, for I could obtain no deposit on gold, from one-sixteenth of a grain of corrosive sublimate in sixteen ounces of porter. It may be objected that this galvanic process only proves the presence of mercury: but a medical jurist can seldom do more. It would show that mercury was present in some tangible form; and the only common soluble salts of this metal are both highly poisonous—namely, corrosive sublimate and pernitrate:—whether the substance had acted as a poison or not, would be determined from symptoms and post-mortem appearances;—whether it had been given or intended as a medicine or not, would be easily deduced from other circumstances. The only way to prove that the mercury was really in the form of corrosive sublimate, would be either by the discovery of some undissolved portions of the solid poison in the stomach or its contents:—or by a separation of the poison, when actually in solution, by means of ether. The addition of nitrate of silver to the contents of the stomach, or to an article of food, in order to detect the chlorine, would be in the highest degree objectionable; since the nitrate of silver is not only precipitated abundantly by most kinds of organic matter, but also by common salt, which, if not taken as an article of food, is always present naturally in the gastric secretions. Such an experiment might in certain cases be the basis of a chemical suspicion of the nature of the mercurial compound, but not of a medico-legal opinion.

Let us suppose that the filtered liquid contains no trace of a mercurial salt, we must now direct our attention to the analysis of the *insoluble matters* separated by filtration. These may be boiled in distilled water; the liquid filtered and tried by ether; if this does not succeed, it may be tried by the galvanic test. I have found that most of the compounds which corrosive sublimate forms with organic matter, yield commonly sufficient poison for detection by boiling them in water, when the analysis has not long been delayed. But supposing that the solid matters,—even comprising the stomach itself, cut into pieces,—yield no traces of the poison, it will be difficult to obtain evidence of its presence;—the process to be presently described for detecting mercury in the organic tissues, may then be resorted to.

Dr. Christison has recommended the use of protochloride of tin as a precipitant in organic mixtures. If this be used, the precipitate should be well boiled in strong muriatic acid, and afterwards in potash, in order to separate from it any oxide of tin and organic matter. This process is indispensable, when we wish to ascertain the total quantity of mercury present, so that the quantity of corrosive sublimate may be determined. When the protochloride of tin does not give a precipitate in a suspected liquid, it is pretty certain that no mercury will be detected either by the galvanic or any other test. When the quantity of poison is small, I have found that there is much trouble and some risk of loss in attempting to separate the mercury by the protochloride of tin, and that it cannot be readily done. By means of the galvanic test, I was enabled to detect the one-sixteenth part of a grain of corrosive sublimate dissolved in one ounce of an organic liquid, and to obtain the metallic mercury from it in less than half an hour. The deposition on the gold was slow, but the mercury was entirely accumulated on the metal. Protochloride of tin gave an abundant precipitate with the same proportion of poison, but it was a much more troublesome and tedious process to obtain the mercury from this precipitate.

It is not possible to conceive a case where, in an analysis of this kind, the galvanic test would not be immediately applicable. This, it will be observed, is merely the medium for furnishing a ring of pure metallic mercury; and as in the case of arsenic, it is not so much from the *quantity* of arsenic we obtain by the reduction-process, as from the clear and undoubted evidence of its presence, in the physical and other properties of the metal;—so in the case of corrosive sublimate, it is not necessary to precipitate *all* the mercury in a given organic mixture, in order to say that this metal is there present. Thus, then, if from an organic liquid, by means of the galvanic test, we can obtain a distinct sublimate of mercury, we need not employ either the protochloride of tin or sulphuretted hydrogen gas, to separate the whole of the poison, unless our object be to determine the total quantity present. An arsenical sublimate weighing less than the 1-50th part of a grain, furnishes evidence of the presence of that metal; the same is true of mercury, however the sublimate may be procured; and an operator has no more occasion to obtain all the mercury from the corrosive sublimate contained in a stomach, than he has to obtain all the arsenic from the arsenious acid diffused through the stomach and intestines in a case of arsenical poisoning.

The poison not detected.—Corrosive sublimate is not easily lost in organic liquids, when it is in moderate quantity. A few grains of this poison were mixed with some ounces of albumen, gruel, and porter, in January 1839. An abundant precipitate was formed. The mixture has been loosely exposed for eight years: but the poison is now easily discovered by the protochloride of tin, by the galvanic test, and by metallic silver, both in the supernatant liquid and in the precipitate. Corrosive sublimate is not always found in the stomachs of persons poisoned by it, although, from its readily combining with the mucous membrane, it is more likely to be detected than arsenic. In a well-marked case, which occurred to Mr. Watson, where two drachms killed a person in six days, none was found on a chemical analysis of the contents. This may have been partly due to the length of time that had elapsed. In a case in which two drachms were swallowed, and the man died in four days, no mercury was detected in the stomach or tissues, (p. 322, ante.) In Mr. Illingworth's case, where probably less was taken, and death occurred rapidly, it was freely detected in a state of solution, (p. 327, ante.)

If a person has died under symptoms of mercurial poisoning, it will generally be sufficient that the chemical analysis should show that mercury was present in the *contents* of the viscera. In what form the mercury was taken, is a fact which must be proved by other circumstances: but it may be objected, whenever mercury is found in an insoluble state in the viscera or their contents, that it had been derived from some mercurial preparation administered medicinally, such as calomel, blue pill, or grey powder. This must be admitted, as the specific poison is not, in such cases, readily susceptible of detection; but the very obvious answer is, that symptoms and post-mortem appearances indicate the fact of poisoning, and the analysis is merely corroborative. The discovery of a *soluble* compound of mercury in any suspected organic liquid, whether administered or not, must always remove the suspicion of its being derived from a medicinal preparation: if corrosive sublimate have itself been used as a medicine, it will be sufficiently apparent from circumstances. Besides, the dose in such cases is always extremely small. (See p. 345.)

Detection of absorbed mercury in the tissues.—The researches of numerous chemists, as well as the more recent experiments of MM. Danger and Flandin, have removed any doubt which might have formerly existed, respecting the *absorption* of mercury in cases of poisoning. It is said that corrosive sublimate is itself absorbed, but I am not aware that there is any proof of this,—all we know is, that *mercury* has been extracted from the tissues of the body. The experimentalists above mentioned, a few years since presented a memoir to the Academy of Sciences, detailing their method of detecting absorbed mercury. They heat the animal matter, finely cut up, to about 212°, with one-third or one-half of its weight of strong sulphuric acid. In an hour or two, the whole forms a dark carbonaceous-looking liquid. It is allowed to cool, and powdered chloride of lime is then gradually added. The liquid becomes whiter and more viscid. The quantity of chloride used is about equal to the weight of sulphuric acid: it is added until the whole appears like a white calcareous mass. The residue dried is then digested in absolute alcohol, which dissolves the mercurial compound; it is now diluted with distilled water, and the earthy residue is repeatedly washed,—the liquids being afterwards mixed and concentrated. The concentrated liquid is placed in a funnel terminating at an angle of 90° in a capillary point,—the galvanic plates of gold and tin being introduced into the contracted part of the funnel. In this way every drop of liquid comes in contact with the metals, and the gold is slowly covered with the mercury. It is stated that the metal has thus been detected in a solution containing the 100,000th part, which shows that the poison may be exceedingly diluted and yet susceptible of detection. These gentlemen have thus extracted mercury from rather more than three ounces of the liver of an animal poisoned by corrosive sublimate. What absolute quantity of the poison they have succeeded in detecting in their experiments is not stated. (See Comptes Rendus, 31 Mars, 1845, 951; also, Ann. d'Hyg. 1846, i. 206.) This is obviously nothing more than the old galvanic process, modified by allowing each particle of liquid to come slowly in contact with the metallic plates. The use of the chloride of lime does not appear to be absolutely necessary, and it materially adds to the bulk of the substance for analysis.

The diffusion of this poison through the system even when mercurial preparations have been applied externally, is well illustrated by the fact that Landerer detected mercury in the pus from the bubo of a man who had employed mercurial frictions. (Heller's Archiv. 1847, H. 2, p. 185.)

Any of the processes elsewhere described (see chapter on ANALYSIS, ante, p. 132,) may be employed. We may bring the organic matter to a state of perfect solution by muriatic acid, we may carbonize it by sulphuric acid, or we may entirely destroy it by nitro-muriatic acid. The surplus acid, in each case, must be driven off or neutralized by an alkali before copper gauze or the plates of gold and zinc are used. Orfila states that in boiling a solution of corrosive sublimate, a portion of the poison is lost by evaporation with the water. (For a method of preventing any loss, see p. 295, ante.)

In solids.—Corrosive sublimate may be easily detected in organic *solids* by boiling a small portion of the solid with copper gauze and a few drops of muriatic acid; or the plates of gold and zinc may be at once used. I have thus in one instance detected it in snuff.

QUANTITATIVE ANALYSIS.—If the poison be entirely in a soluble form, we may procure the mercury from a part only by the protochloride of tin, and calculate the remainder proportionably. If it be in an insoluble form, we must then pursue the process recommended in the preceding page, and precipitate it entirely by the salt of tin, purifying the mercury by boiling it first in potash, and secondly in muriatic acid. For every 100 grains of metallic mercury obtained, we must allow 135 grains of crystallized corrosive sublimate to have been present.

CALOMEL.

This substance, now called chloride of mercury, although commonly regarded as a mild medicine, is capable of destroying life, even in comparatively small doses. Several cases have been already referred to, where excessive salivation, gangrene of the salivary organs, and death, have followed from the medicinal doses of a few grains, (p. 312.) There is a case reported in the Medical Gazette (xviii. 484,) in which a boy, aged fourteen, was killed in about three weeks by a dose of only *six grains* of calomel. It is singular that in this case neither the teeth nor the salivary glands were affected; still, considering the effects of calomel in other instances, it seems most probable, that the ulceration and gangrene of the face, which followed, were due to it. Pereira mentions the case of a lady who was killed by a dose of twenty grains of calomel; she had previously taken a moderate dose without a sufficient effect being produced.

Sobernheim states that a girl, aged eleven, took in twenty-four hours eight grains of calomel, for an attack of tracheitis, and died in eight days from inflammation and ulceration of the mouth and fauces. In another instance, which occurred to Lesser, fifteen grains of calomel produced similar effects, with excessive salivation; and this patient also died in eight days. Meckel relates that twelve grains have destroyed life. (Lehrbuch der Ger. Med., 267.) Two cases of death from calomel, in children, are recorded in the Registration returns for 1840.

There are many other fatal cases on record, and the facts seem to leave no doubt that calomel may, in large doses, act as an irritant poison. It was supposed that these effects might be ascribed to this compound being adulterated with corrosive sublimate; but Dr. Christison examined ten different specimens of calomel without finding in one so much as one five-hundredth part of its weight of corrosive sublimate; this would be less than a grain to an ounce; and in a common dose of three grains of calomel there would be no more than the one hundred and sixtieth part of a grain of corrosive sublimate,—a quantity insufficient to do mischief. (Ed. Med. and Surg. Journ. xlix. 336.) It has been further supposed that calomel might be converted into corrosive sublimate, by the free muriatic acid contained in the gastric secretions; but the very minute proportion in which this acid exists in the gastric juice, according to Dr. Prout, renders this explanation improbable.

With these facts before us, it is a remarkable circumstance that calomel has been often taken in very large doses medicinally and accidentally, without producing dangerous consequences. Dr. Pereira states that upwards of five ounces of calomel were given in forty-two hours, to one cholera patient, without producing any sensible effect. (Med. Gaz. xviii. 468.) It may be objected, however, that we are hardly entitled to draw a fair inference of its effects on a healthy person, from exhibiting it in cases of disease; since, as it is well-known, opium itself may be given in poisonous doses, to persons affected with certain diseases, without any injury resulting. The following case, however, shows, that even *an ounce* may be taken by a healthy person without serious effects resulting. A healthy girl, aged nineteen, swallowed about an ounce of calomel by mistake for magnesia. She took it in milk, and rinsed out the cup, so that the whole was swallowed except a few grains. The mistake was not discovered until two hours afterwards: she then experienced slight nausea and faintness. Emetics and lime-water were given, and in about half an hour the larger portion of the calomel was discharged, mixed with mucus. Severe griping pains with tenderness of the abdomen came on; but in the course of four days the girl had completely recovered without the salivary organs having become in the least degree affected. (Med. Gaz., July 1838.) It is proper that a medical jurist should know that life has been destroyed by this medicine; and that death from calomel has become a subject of medico-legal investigation. On these occasions it is likely there will be some difference of opinion respecting the cause of death. An interesting case of this kind, in which a man was tried for the murder of his wife by calomel, is related by Dr. Christison. (Op. cit. 430.) Mr. Swan found that calomel given to a full-sized dog, in doses of from three to four grains night and morning for a week, gave rise to ulceration of the gums, a discharge of bloody saliva, loss of power in the hinder legs, and death on the ninth day. The stomach and all the thoracic and abdominal viscera were sound, with the exception of some appearances of chronic inflammation in the large intestines. A similar experiment on another dog gave like results, and in both cases the ganglia of the sympathetic nerves were found inflamed. (Action of Mercury, p. 5. 1847.) These, with the facts above related, show how erroneous it is to suppose, that because a body is quite insoluble in water, it is therefore inert. A medical jurist must bear in mind that, under certain diseased states of the body, a simple medicinal dose of calomel may produce the most serious effects on young subjects, and even cause death. (See *Salivation*, ante p. 312.)

Conversion of calomel to corrosive sublimate by alkaline chlorides.—There are various ways in which corrosive sublimate in solution may become partially decomposed and converted into calomel. Long exposure to light, especially of the alcoholic solution, is sufficient for this purpose. On the other hand, it is important to inquire, in relation to a substance so extensively used for medicinal purposes, whether in some instances calomel may not become partly converted to corrosive sublimate within the body, and act as a poison. A case of some interest in relation to this point occurred recently in France. A physician prescribed for a child, powders, containing in each, five grains of muriate of ammonia, five grains of sugar, and half a grain of calomel. After taking a few of these powders, the child died with all the symptoms of poisoning by corrosive sublimate; and the apothecary was charged with the death of the child, on the supposition that he had substituted corrosive sublimate for calomel. Experiments were instituted to ascertain whether the calomel could have been decomposed by the ammoniacal salt; and it was satisfactorily proved, that a portion at least was converted into corrosive sublimate. M. Mialhe, who reports the case, has confirmed this result. He states that calomel, when mixed with muriate of ammonia,—the chlorides of sodium (common salt,) or potassium and distilled water, is partly transformed into corrosive sublimate and metallic mercury. He found that this decomposition took place at the temperature of the body, and even at the ordinary temperature, (60°) in the course of a few seconds. The mercurial or metallic taste of calomel depends, according to this gentleman, on its partial conversion into corrosive sublimate by the alkaline chlorides contained in the saliva; and the quantity of sublimate formed within the body on the taking of calomel, will, therefore, depend on the quantity of alkaline chloride (salt) which it may meet with in the alimentary canal. (Ed. Med. and Surg. Journ., July 1840.) These facts are of some importance in relation to the administration of calomel as a medicine, more especially in conjunction with alkaline chlorides, or where much salt is introduced into the stomach as an article of food.

The following are the results of some experiments which I have made in respect to this alleged conversion of calomel into corrosive sublimate. A few grains of calomel were boiled for some minutes in a saturated solution of common salt; it became of a dark grey colour, and when the filtered liquid was tested, it gave the usual reactions indicative of the presence of a portion of corrosive sublimate. The solution was precipitated of a scarlet colour by iodide of potassium, which, as we have seen, is one of the weakest of the tests for this poison:—but at common temperatures the effects were widely different. 1. Fifteen grains of calomel were digested for two months in an ounce of diluted muriatic acid. The liquid when filtered gave, with protochloride of tin, a very faint cloudiness,—but not the slightest deposit of mercury took place in twenty-four hours, on introducing gold and zinc. 2. The same quantity of calomel was introduced into one ounce of a saturated solution of common salt:—after two months' digestion protochloride of tin gave, with about one-fourth of the liquid, a dark-grey turbidness:—but by this means, no mercury could be procured. A piece of gold with zinc became, however, slightly coated in twenty-four hours, but iodide of potassium had no effect. This showed that only a very minute portion of corrosive sublimate could have been produced. 3. A mixture was made of ten grains of muriate of ammonia, ten grains of sugar, and one grain of calomel, in one ounce of water. After a lapse of two months there was not sufficient corrosive sublimate formed, to conceal, by a deposition of mercury, the yellow colour of the gold, although the coil of gold and zinc remained in the liquid twenty-four hours. Here the quantities were twice as great as those contained in the powder. It might be objected that this change may be materially affected by temperature. In the above experiments the temperature had been about 55°:—but in another case, 4, a similar mixture, was kept for two hours by means of a water-bath at about 98°, and the liquid was filtered and tested. Protochloride of tin rendered a portion of it turbid: a slip of copper was faintly coated with mercury in twenty-four hours, but the yellow colour of gold was not entirely concealed on the application of the galvanic test. 5. In mixing the above materials with a quantity of water at 60°, sufficient to form a kind of paste, the powder became grey in a few seconds, clearly indicating that the calomel was undergoing a partial conversion into corrosive sublimate. Admitting, therefore, that a change of this kind does take place, it seems to me very improbable, that the quantity of corrosive sublimate formed from a few grains of calomel, should be sufficient to produce symptoms of irritant poisoning, or to cause death. Nevertheless, a case has been recently reported in which death was referred to this cause. A man affected with cerebral fever was ordered occasional doses of calomel with an enema composed of decoction of senna and a table-spoonful of common salt. The patient died the same evening, and death was ascribed to the formation of corrosive sublimate in the body by the chemical reactions of the two salts. (Journal de Pharmacie, Janvier 1846.) This case is very imperfectly reported. There is no satisfactory account of symptoms or post-mortem appearances, nor is it alleged that corrosive sublimate was found in the dead body. Besides it is not easy to understand how the common salt given by the rectum should produce corrosive sublimate with the calomel taken into the stomach!

It has been already remarked that calomel itself is often mixed with corrosive sublimate. On boiling one grain in distilled water, filtering the liquid, and applying the tests for corrosive sublimate, more especially the protochloride of tin, effects very similar to those just described, although less in degree, were produced.

CHEMICAL ANALYSIS.—Calomel is commonly seen in the form of a white powder with a faint yellow tint, so insoluble in water, that it is said to require 12,000 parts to dissolve one part. It is also insoluble in alcohol and ether, which may be thus usefully employed to separate from and detect in it any traces of corrosive sublimate. Calomel, when long exposed to light, becomes deeper in colour; this is thought by some to be owing to its partial conversion to corrosive sublimate; but there is no evidence of any of this poisonous salt being thus produced. The specific gravity of calomel is much greater than that of corrosive sublimate; and owing to this property it is not difficult to separate it from organic liquids by decantation. Among the characters by which calomel may be identified, we may note the following:—1. Its insolubility in water;—it is soluble in strong nitric acid, but decomposed by it into corrosive sublimate and pernitrate of mercury. Strong muriatic acid transforms it to corrosive sublimate and metallic mercury; and nitro-muriatic acid converts it readily to corrosive sublimate on boiling. 2. When heated on platina over a spirit-lamp it is sublimed, but it is not so volatile as corrosive sublimate: during sublimation it is partially decomposed into that substance and metallic mercury. 3. When dropped into a solution of iodide of potassium, it is slowly turned of a dingy, greenish-black colour; but if the iodide be much diluted, the powder acquires a yellow colour. 4. By a solution of potash, it is turned black, chloride of potassium being formed in the liquid. 5. It is also turned black by a solution of hydrosulphuret of ammonia. 6. It gives a silvery stain when rubbed on clean copper with diluted muriatic acid. 7. It yields a ring of metallic mercury when heated with the carbonate of soda. 8. It is decomposed by a solution of protochloride of tin, and reduced to metallic mercury. In some of these characters it resembles corrosive sublimate, but it is eminently distinguished from that body by the first, third, and fourth. In addition to these differences, calomel is turned black by solution of ammonia, while corrosive sublimate forms a white compound. In order to detect the chlorine, it is necessary to boil in water the residue, left in the reduction-tube after complete sublimation of the mercury: and then add to the filtered liquid, neutralized by nitric acid, nitrate of silver.

WHITE PRECIPITATE. AMMONIO-CHLORIDE OF MERCURY.

This is an irritant compound, although little is known concerning its effects. In January, 1840, a young woman, who had swallowed this substance, was received into St. Thomas's Hospital. She had mixed it up and taken it in water,—but the quantity swallowed could not be ascertained. The stomach-pump was employed, mucilaginous drinks and olive-oil were administered; and in the course of a few days she perfectly recovered. The symptoms under which she suffered were those of gastric irritation. Judging from this case, white precipitate does not appear to be a very active preparation. One instance of death from salivation produced by this compound, is recorded in the Registration returns for 1840, in a child, aged seven.

Chemical analysis.—This powder resembles corrosive sublimate in being entirely volatilized by a moderate heat, and in giving a metallic sublimate with dried carbonate of soda, but it differs in being insoluble in water. By heating it with solution of potash, ammonia is evolved, chloride of potassium formed, whereby the chlorine may be detected, and yellow peroxide of mercury, after long boiling, is left, which may be easily analyzed in the way described at p. 330.

BLACK OXIDE OF MERCURY.

This appears to be one of the least injurious preparations of mercury. It is a purgative, and in large doses would probably act as an irritant. In small doses frequently repeated, it gives rise to salivation and other symptoms indicative of the action of mercury. I have not been able to find any case in which it has produced injurious consequences in the human body.

Chemical analysis.—It is a compound of a black, or if long kept (owing to partial decomposition) of a greenish colour. 1. It has no taste, and is insoluble in water and alkalies. 2. Either nitric or acetic acid will dissolve it. 3. Muriatic acid produces with it, calomel. 4. When heated it gives off oxygen, and a mercurial sublimate is obtained. The only case in which this compound is liable to give rise to a question of poisoning is in reference to its use in *Black wash*—made by mixing lime water and calomel. This compound is formed of black oxide of mercury and chloride of calcium: it is used only externally, but by accident it may be swallowed. The oxide is supposed to exist in some medicinal compounds, such as blue pill, mercurial ointment, and grey powder or mercury and chalk. The more probable view, however, is, that the mercury is in these cases only in a very fine state of mechanical division.

BLUE PILL.

The account of an inquest on a person alleged to have died from the effects of *Blue pill*, will be found reported in the Medical Gazette, October 1843. It appears that the deceased, æt. 40, took some medicine prescribed for him by a practitioner. It consisted of six grains of blue pill and three of calomel. This was alleged to have produced salivation and a mercurial fever, of which the man died in about seven weeks. The salivation was probably owing to a remarkable idiosyncrasy; even a smaller dose than that here prescribed, has been known to cause fatal salivation. But from the evidence, it was not improbable that the deceased had taken some quack pills, which, had their composition been known, might have accounted for the severity of the symptoms. The jury returned a verdict of natural death, but called the remedy administered "an overdose of strong medicine." (Ph. Journ., Nov. 1843.) This compound can hardly be considered as an active poison. In doses of from five to ten grains it acts as a purgative. The only cases in which it has produced serious or fatal effects are those in which excessive salivation has followed its long-continued use in small doses.

Chemical analysis.—The odour and colour are sufficient to identify this compound. No globules of mercury can be seen if it be well prepared, but when rubbed on a clean surface of gold, it gives a white mercurial stain. By nitro-muriatic acid, the organic matter is decomposed and the mercury converted to corrosive sublimate. In this way it may be analyzed. Three grains of blue-pill contain one grain of mercury.

MERCURIAL OINTMENT. UNGUENTUM HYDRARGYRI.

This is a mixture of finely-divided mercury in a metallic state with lard and suet. It is of a blueish-grey colour, commonly used externally, and producing salivation and other mercurial symptoms by absorption. It is not often given internally, although its effects would be doubtless the same as those of blue pill. I lately had to examine many articles found in the possession of a woman charged with the crime of poisoning. There were several specimens of this ointment among them; but there was no proof that it had been administered as a poison. It would require a very large dose to produce any serious effects, and from its nauseous character it would probably be rejected by the stomach.

Chemical analysis.—Its colour and physical properties will serve to identify it. When well prepared no globules are visible to the eye, but it tarnishes with a mercurial stain a polished surface of gold. The fat may be separated by boiling in water: the particles of mercury subside, and any adhering portions of fat may be removed by digestion in ether or oil of turpentine. Two drachms of this ointment (Ung. Hyd. Fort.) contain one drachm of mercury. There is a more diluted preparation (Ung. Hyd. Mitius;) six drachms of this contain one drachm of mercury.

GREY POWDER. MERCURY WITH CHALK.

This a compound of carbonate of lime and finely-divided mercury. It is an innocent preparation, and acts as a purgative on children in doses of from three to five grains, and on adults in doses of from five to fifteen grains. It would require a very large dose to produce any injurious effects, and it is here enumerated among mercurial preparations because poisonous effects might be improperly ascribed to it. Its use long continued in small doses, produces the usual mercurial symptoms.

Chemical analysis.—It is of a greyish-blue colour, except when long exposed to light and heat, whereby it becomes lighter. When heated it yields a mercurial sublimate, and leaves carbonate of lime. Acetic acid dissolves the lime and leaves the mercury: nitric acid dissolves both. Globules of mercury are commonly visible in this powder by the aid of a lens. Eight grains of the powder contain three grains of mercury.

RED PRECIPITATE. RED OXIDE OF MERCURY.

This substance is commonly met with in crystalline scales of various shades of colour, from a dusky to a bright red, and forming an orange-coloured powder. It is poisonous, but instances of poisoning by it are very rare. The following case occurred at Guy's Hospital in 1833. A woman aged twenty-two, who had swallowed a quantity of red precipitate, was brought in labouring under the following symptoms. The surface was cold and clammy,—there was stupor approaching to narcotism,—frothy discharge from the mouth, and occasional vomiting:—the vomited matters contained some red powder which was proved to be red precipitate. There was considerable pain in the abdomen, increased by pressure; and there were cramps in the lower extremities. On the following day, the fauces and mouth became painful, and the woman complained of a coppery taste. The treatment consisted in the use of the stomach-pump, and the free administration of albumen and gluten. She left the hospital four days afterwards, still under the influence of mercury. The quantity of oxide here taken, was not ascertained.

Sobernheim reports a case, where a man, aged twenty-six, swallowed an ounce of red precipitate. He was speedily attacked with pain in the abdomen, nausea, purging, cramps and general weakness. The vomited matters consisted of masses of mucus, containing red precipitate. He continued to get worse, and died in less than forty-eight hours after taking the poison. On inspection, the mucous membrane was found eroded and inflamed in patches, small particles of the poison being imbedded in it. The duodenum was in a similar state, and there was a large quantity of red precipitate in the contents of the viscus, as well as in the stomach. (Op. cit. 250.) Niemann quotes a case in which six grains caused the death of a woman. (Taschenb. der Arzneiw. 452.) Notwithstanding the fatal result in these cases, red precipitate does not appear to be a very active irritant poison. *Thirty grains* of it have been taken and retained in the system, with comparative impunity. Some years ago a young woman swallowed this quantity, with a suicidal intention. Almost immediately afterwards, emetics of ipecacuanha and sulphate of zinc were administered, and the stomach-pump was used repeatedly; but none of the red powder was ejected. She suffered chiefly from pain in the abdomen and general weakness, but in the course of a few days she recovered. Devergie relates another instance of recovery in which a much larger dose had probably been taken. (Méd. Lég. ii. 705.)

A common opinion exists among the vulgar, that this compound is possessed of very active poisonous properties: hence it is sometimes administered with criminal design. At the Chester Lent Assizes, 1846, a woman was charged with attempting to poison a child by means of this substance. It was proved in evidence that the prisoner had given to a child, aged two months, about a teaspoonful of the nitric oxide. Vomiting of a reddish liquid speedily ensued; the greater part of the poison appears to have been thrown off the stomach, and the child recovered. The prisoner was convicted of the offence. The chemical evidence clearly established the nature of the compound; but a question arose whether it was to be considered a *deadly* poison or not, (see ante, page 16.) It has no claim to be so considered. (Med. Gaz. xxxvii. 874.)

The *Yellow wash*, made with a mixture of corrosive sublimate and lime water, owes its poisonous properties to precipitated red oxide. If the corrosive sublimate be in excess it may be an oxychloride. This lotion being in common use may easily give rise to accidents.

The nitric oxide has a local action as a stimulant, irritant, and escharotic. The ointment commonly used (Ung. Hyd. Nit. Oxydi) contains one-ninth part by weight of the oxide.

Chemical analysis.—Red precipitate is known,—1. By its being in red crystalline scales. 2. By its insolubility in water,—this, together with its great weight, renders it easy of separation from *organic liquids*. 3. It is readily dissolved by warm muriatic acid, forming a solution possessing all the properties of corrosive sublimate. 5. When heated in a small tube, it becomes black (reacquiring its red colour on cooling;) and while an abundant sublimate of metallic mercury is formed, oxygen gas is evolved. If the heat be continued, it should be entirely dissipated when pure,—a property by which it is known from most other red powders. In this experiment, a slight yellow sublimate is sometimes formed (subnitrate,) owing to the oxide retaining some traces of nitric acid.

IODIDES OF MERCURY.

The yellow and the scarlet iodides of mercury are irritant poisons which, judging from their effects on animals, are likely to act powerfully as irritants in the human subject. They have also a local action. They are quite insoluble in water, and both yield a mercurial sublimate when heated with carbonate of soda. The scarlet iodide is further known by its becoming yellow when heated, and again red on trituration.

CINNABAR. VERMILION. PERSULPHURET OF MERCURY.

The term *Cinnabar* is applied to a dark and heavy compound of sulphur and mercury, while *Vermilion* is the same substance reduced to a fine powder. It is well known as a red pigment, and is often employed in colouring confectionary, wafers, &c. I have not been able to find any instance of its having acted as a poison on man. Orfila believes that it is not poisonous. It has, however, proved fatal to animals in the proportion of from thirty to seventy grains, even when applied externally to a wound. Cinnabar is sometimes used for giving a red colour to ointments, *e. g.* the sulphur ointment. In such cases the quantity is very small, and can do no injury even if swallowed.

Dr. Sutro has published a short abstract of a case in which the *vapour* of *Vermilion* applied externally produced severe symptoms. A woman, by the advice of a quack, applied this vapour to a cancerous breast. She employed three drachms of vermilion, covering herself with a sheet so that the vapour should only reach the body externally. After three fumigations, she suffered from severe salivation and violent fever, which continued for four weeks. The right arm became œdematous. (Med. Times, Sept. 27, 1845, p. 17.)

Chemical analysis.—Vermilion is of a rich red colour, very heavy, and quite insoluble in water. When dropped into the hydrosulphuret of ammonia its colour remains unchanged; while red precipitate and red lead are turned of a dark brown colour, or even black. It is also known from red precipitate by its insolubility in muriatic acid. 1. When heated on platina it is entirely volatilized, the sulphur burning away. 2. Heated in a reduction-tube with carbonate of soda, a sublimate of metallic mercury is obtained with a residue of sulphuret of sodium, in which sulphur may be easily proved to exist by the usual tests, *e. g.* by placing a portion of the residue on a glazed card and adding a drop of water. (See p. 174, ante.)

BICYANIDE OF MERCURY.

This is a substance which is but very little known, except to chemists, yet it is an active poison, and has caused death in one instance. In April 1823, a person who had swallowed twenty grains of this compound (thirteen decigrammes,) was immediately seized with all the symptoms of poisoning by corrosive sublimate, and died in nine days. There was continued vomiting with excessive salivation, ulceration of the mouth and fauces, suppression of urine, purging, and, lastly, convulsions of the extremities. On inspection, the mucous membrane of the stomach and intestinal canal, was extensively inflamed. (Orfila, i. 583.) Dr. Christison quotes a case in which ten grains destroyed life within the same period of time. (On Poisons, 427.) As a poison, the bicyanide is probably not much inferior in activity to the bichloride of mercury.

Chemical analysis.—This is a white salt commonly crystallized in quadrangular prisms. It is very soluble in water, especially at the boiling point. The solution has no odour of prussic acid until an acid is added. Its solution is neutral, and is not precipitated by caustic potash. 1. If the powdered crystals be heated in a small tube, cyanogen gas is evolved and may be burnt at the mouth, the flame being of a rich rose-red colour, with a blue halo:—metallic mercury is at the same time sublimed. This experiment is alone sufficient to identify the poison. The solution of bicyanide is not unlikely to be mistaken for that of corrosive sublimate. It readily deposits mercury on copper and gold by the galvanic test, and it is precipitated black by hydrosulphuret of ammonia and sulphuretted hydrogen gas. It is known, 1. By potash producing in it no precipitate; 2. By prussic acid being evolved on boiling a portion of it with muriatic acid, corrosive sublimate being produced in the liquid; 3. By a scarlet precipitate being formed on adding to the solution, iodide of potassium and muriatic acid.

TURBITH MINERAL. SUBSULPHATE OF MERCURY.

Fatal cases of poisoning by this compound are by no means common. It is undoubtedly, although very insoluble, a strong irritant poison, and is capable of causing death in a comparatively small dose. A well-marked instance of its fatal operation was communicated to the Pathological Society by Mr. Ward in March 1847. A boy, æt. 16, swallowed *one drachm* of this preparation on the night of February 19th. It produced a burning sensation in the mouth and throat, and vomiting in ten minutes. In about an hour there was paleness with anxiety of countenance, coldness of surface, constant sickness, sense of heat and constriction in the throat, and burning pain in the stomach with cramps. The irritability of the stomach continued in spite of treatment, and after two days, there was salivation with mercurial fœtor. The gums acquired a deep blueish tint and began to ulcerate. The patient died in about a week after he had taken the poison, without convulsions, and without suffering at any period from symptoms of cerebral disturbance. The principal post-mortem appearances were,—inflammation of the œsophagus; its mucous membrane at the lower part peeling off;—the inner surface of the stomach near the cardia and pylorous, was covered with petechial spots;—the small intestines were contracted, the inner coat reddened and petechial spots were found, but chiefly in the large intestines. The parotid and submaxillary glands were swollen. Mercury was detected in the intestines (see Med. Gaz. xxxix. 474.) From this account, it will be perceived that turbith mineral has an action somewhat similar to corrosive sublimate, although it is probably much less active.

Chemical analysis.—Turbith mineral is a heavy powder of a yellow colour, becoming of a dark olive by exposure to light. It is scarcely soluble in water, but has a strong metallic taste. When heated in a tube, with or without carbonate of soda, it yields metallic mercury. It is best analyzed by boiling it in potash, in which case sulphate of potash and peroxide of mercury result—the acid and the base are then easily determined.

NITRATES OF MERCURY.

These are corrosive poisons which are used for several purposes in the arts. They are solid white salts, easily dissolved by water, especially if there be a little excess of acid present. The acid pernitrate has already caused death in an interesting case reported by Mr. Bigsley in the Medical Gazette (vi. 329.) A butcher's boy dissolved some mercury in strong nitric acid, and swallowed about a teaspoonful of the solution. Soon afterwards he suffered the most excruciating pain in the pharynx, œsophagus and stomach:—there was great anxiety, with cold skin, small pulse, colic and purging. He became gradually weaker, and died in about two hours and a half. On inspection the fauces, œsophagus and stomach, were found corroded and inflamed. Although he survived so short a time, the mucous membrane of the stomach was of a deep red colour.

The solution of acid pernitrate has been used as a cautery, and when diluted, as a lotion in diseases of the skin. Under these circumstances, it has been known to cause severe salivation, sloughing, and suppression of urine. (See p. 320.) A medical practitioner in France, was lately charged with malapraxis, in having improperly prescribed this solution as a wash for the itch. The skin of the body where the lotion had been employed, was discoloured and partly corroded; there was also pain in the throat, difficulty of swallowing, inflamed gums and salivation. The parties recovered from these effects in the course of a short time, and the charge was dismissed on the ground, that the lotion had been improperly used by the complainants; they had used it in a concentrated state, instead of diluting it with water, as ordered by their medical attendant. (Ann. d'Hyg. Juillet 1842.) A case of death from this compound has been recently reported (Journal de Chimie Méd. 1846, p. 734.) There can be no doubt, that this poison is quite as formidable, both as a corrosive and irritant, as corrosive sublimate itself. The diluted protochloride of tin, if timely applied, will counteract the local effects of these corrosive compounds of mercury. The nitrate of mercury is used in the form of ointment (Ung. Hydr. Nitratis) as a local application. A dose of this might exert an irritant action. About ten parts of the ointment contain three of the nitrate.

Chemical analysis.—In the solid state, the crystals of acid *Pernitrate*, when heated in a tube, yield nitrous acid vapour,—peroxide of mercury, and a ring of metallic mercury,—when heated with carbonate of soda, metallic mercury is easily obtained. In *solution*, it is commonly met with in the state of a highly acid liquid, and nitric acid may be obtained by distillation, a result which does not occur with a mere solution of the crystals in water. The solution possesses all the properties of corrosive sublimate, so far as the tests for mercury are concerned; but it gives no precipitate with nitrate of silver. On adding carbonate of potash and filtering, nitrate of potash is easily detected in the filtered liquid, and thus the acid is identified. On evaporating to dryness, and obtaining the solid crystals, they may be easily known from those of corrosive sublimate, by the simple application of heat. The *Protonitrate* is known in solution, both from pernitrate and corrosive sublimate, in its being precipitated black by potash, and white by muriatic acid. This acid does not precipitate the solutions of either of the salts above mentioned.

ACETATE OF MERCURY.

The Acetate is another soluble salt of mercury, but nothing is known respecting it as a poison.

Proportion of Mercury in Pharmaceutical Preparations.—It may be proper to insert in this place the proportion in which mercury enters into various medicinal compounds belonging to the London Pharmacopœia, some of which, by causing death, have given rise to important medico-legal inquiries. Mercury with chalk (Hydrargyrum cum Creta) contains three grains of mercury in eight grains of powder. Blue pill (Pilulæ hydrargyri) contains one grain of mercury in three grains. Compound calomel pill (Pill. hyd. chlor. comp.) contains one grain of calomel in five grains. Iodide of mercury pill (Pill. hyd. iod.) contains one grain of the iodide in five grains. Solution of tially decomposed and converted into calomel. Long exposure to light, especially of the alcoholic solution, is sufficient for this purpose. On the other hand, it is important to inquire, in relation to a substance so extensively used for medicinal purposes, whether in some instances calomel may not become partly converted to corrosive sublimate within the body, and act as a poison. A case of some interest in relation to this point occurred recently in France. A physician prescribed for a child, powders, containing in each, five grains of muriate of ammonia, five grains of sugar, and half a grain of calomel. After taking a few of these powders, the child died with all the symptoms of poisoning by corrosive sublimate; and the apothecary was charged with the death of the child, on the supposition that he had substituted corrosive sublimate for calomel. Experiments were instituted to ascertain whether the calomel could have been decomposed by the ammoniacal salt; and it was satisfactorily proved, that a portion at least was converted into corrosive sublimate. M. Mialhe, who reports the case, has confirmed this result. He states that calomel, when mixed with muriate of ammonia,—the chlorides of sodium (common salt,) or potassium and distilled water, is partly transformed into corrosive sublimate and metallic mercury. He found that this decomposition took place at the temperature of the body, and even at the ordinary temperature, (60°) in the course of a few seconds. The mercurial or metallic taste of calomel depends, according to this gentleman, on its partial conversion into corrosive sublimate by the alkaline chlorides contained in the saliva; and the quantity of sublimate formed within the body on the taking of calomel, will, therefore, depend on the quantity of alkaline chloride (salt) which it may meet with in the alimentary canal. (Ed. Med. and Surg. Journ., July 1840.) These facts are of some importance in relation to the administration of calomel as a medicine, more especially in conjunction with alkaline chlorides, or where much salt is introduced into the stomach as an article of food.

The following are the results of some experiments which I have made in respect to this alleged conversion of calomel into corrosive sublimate. A few grains of calomel were boiled for some minutes in a saturated solution of common salt; it became of a dark grey colour, and when the filtered liquid was tested, it gave the usual reactions indicative of the presence of a portion of corrosive sublimate. The solution was precipitated of a scarlet colour by iodide of potassium, which, as we have seen, is one of the weakest of the tests for this poison:—but at common temperatures the effects were widely different. 1. Fifteen grains of calomel were digested for two months in an ounce of diluted muriatic acid. The liquid when filtered gave, with protochloride of tin, a very faint cloudiness,—but not the slightest deposit of mercury took place in twenty-four hours, on introducing gold and zinc. 2. The same quantity of calomel was introduced into one ounce of a saturated solution of common salt:—after two months' digestion protochloride of tin gave, with about one-fourth of the liquid, a dark-grey turbidness:—but by this means, no mercury could be procured. A piece of gold with zinc became, however, slightly coated in twenty-four hours, but iodide of potassium had no effect. This showed that only a very minute portion of corrosive sublimate could have been produced. 3. A mixture was made of ten grains of muriate of ammonia, ten grains of sugar, and one grain of calomel, in one ounce of water. After a lapse of two months there was not sufficient corrosive sublimate formed, to conceal, by a deposition of mercury, the yellow colour of the gold, although the coil of gold and zinc remained in the liquid twenty-four hours. Here the quantities were twice as great as those contained in the powder. It might be objected that this change may be materially affected by temperature. In the above experiments the temperature had been about 55°:—but in another case, 4, a similar mixture, was kept for two hours by means of a water-bath at about 98°, and the liquid was filtered and tested. Protochloride of tin rendered a portion of it turbid: a slip of copper was faintly coated with mercury in twenty-four hours, but the yellow colour of gold was not entirely concealed on the application of the galvanic test. 5. In mixing the above materials with a quantity of water at 60°, sufficient to form a kind of paste, the powder became grey in a few seconds, clearly indicating that the calomel was undergoing a partial conversion into corrosive sublimate. Admitting, therefore, that a change of this kind does take place, it seems to me very improbable, that the quantity of corrosive sublimate formed from a few grains of calomel, should be sufficient to produce symptoms of irritant poisoning, or to cause death. Nevertheless, a case has been recently reported in which death was referred to this cause. A man affected with cerebral fever was ordered occasional doses of calomel with an enema composed of decoction of senna and a table-spoonful of common salt. The patient died the same evening, and death was ascribed to the formation of corrosive sublimate in the body by the chemical reactions of the two salts. (Journal de Pharmacie, Janvier 1846.) This case is very imperfectly reported. There is no satisfactory account of symptoms or post-mortem appearances, nor is it alleged that corrosive sublimate was found in the dead body. Besides it is not easy to understand how the common salt given by the rectum should produce corrosive sublimate with the calomel taken into the stomach!

It has been already remarked that calomel itself is often mixed with corrosive sublimate. On boiling one grain in distilled water, filtering the liquid, and applying the tests for corrosive sublimate, more especially the protochloride of tin, effects very similar to those just described, although less in degree, were produced.

Chemical analysis.—Calomel is commonly seen in the form of a white powder with a faint yellow tint, so insoluble in water, that it is said to require 12,000 parts to dissolve one part. It is also insoluble in alcohol and ether, which may be thus usefully employed to separate from and detect in it any traces of corrosive sublimate. Calomel, when long exposed to light, becomes deeper in colour; this is thought by some to be owing to its partial conversion to corrosive sublimate; but there is no evidence of any of this poisonous salt being thus produced. The specific gravity of calomel is much greater than that of corrosive sublimate; and owing to this property it is not difficult to separate it from organic liquids by decantation. Among the characters by which calomel may be identified, we may note the following:—1. Its insolubility in water;—it is soluble in strong nitric acid, but decomposed by it into corrosive sublimate and pernitrate of mercury. Strong muriatic acid transforms it to corrosive sublimate and metallic mercury; and nitro-muriatic acid converts it readily to corrosive sublimate on boiling. 2. When heated on platina over a spirit-lamp it is sublimed, but it is not so volatile as corrosive sublimate: during sublimation it is partially decomposed into that substance and metallic mercury. 3. When dropped into a solution of iodide of potassium, it is slowly turned of a dingy, greenish-black colour; but if the iodide be much diluted, the powder acquires a yellow colour. 4. By a solution of potash, it is turned black, chloride of potassium being formed in the liquid. 5. It is also turned black by a solution of hydrosulphuret of ammonia. 6. It gives a silvery stain when rubbed on clean copper with diluted muriatic acid. 7. It yields a ring of metallic mercury when heated with the carbonate of soda. 8. It is decomposed by a solution of protochloride of tin, and reduced to metallic mercury. In some of these characters it resembles corrosive sublimate, but it is eminently distinguished from that body by the first, third, and fourth. In addition to these differences, calomel is turned black by solution of ammonia, while corrosive sublimate forms a white compound. In order to detect the chlorine, it is necessary to boil in water the residue, left in the reduction-tube after complete sublimation of the mercury: and then add to the filtered liquid, neutralized by nitric acid, nitrate of silver.

WHITE PRECIPITATE. AMMONIO-CHLORIDE OF MERCURY.

This is an irritant compound, although little is known concerning its effects. In January, 1840, a young woman, who had swallowed this substance, was received into St. Thomas's Hospital. She had mixed it up and taken it in water,—but the quantity swallowed could not be ascertained. The stomach-pump was employed, mucilaginous drinks and olive-oil were administered; and in the course of a few days she perfectly recovered. The symptoms under which she suffered were those of gastric irritation. Judging from this case, white precipitate does not appear to be a very active preparation. One instance of death from salivation produced by this compound, is recorded in the Registration returns for 1840, in a child, aged seven.

Chemical analysis.—This powder resembles corrosive sublimate in being entirely volatilized by a moderate heat, and in giving a metallic sublimate with dried carbonate of soda, but it differs in being insoluble in water. By heating it with solution of potash, ammonia is evolved, chloride of potassium formed, whereby the chlorine may be detected, and yellow peroxide of mercury, after long boiling, is left, which may be easily analyzed in the way described at p. 330.

BLACK OXIDE OF MERCURY.

This appears to be one of the least injurious preparations of mercury. It is a purgative, and in large doses would probably act as an irritant. In small doses frequently repeated, it gives rise to salivation and other symptoms indicative of the action of mercury. I have not been able to find any case in which it has produced injurious consequences in the human body.

Chemical analysis.—It is a compound of a black, or if long kept (owing to partial decomposition) of a greenish colour. 1. It has no taste, and is insoluble in water and alkalies. 2. Either nitric or acetic acid will dissolve it. 3. Muriatic acid produces with it, calomel. 4. When heated it gives off oxygen, and a mercurial sublimate is obtained. The only case in which this compound is liable to give rise to a question of poisoning is in reference to its use in *Black wash*—made by mixing lime water and calomel. This compound is formed of black oxide of mercury and chloride of calcium: it is used only externally, but by accident it may be swallowed. The oxide is supposed to exist in some medicinal compounds, such as blue pill, mercurial ointment, and grey powder or mercury and chalk. The more probable view, however, is, that the mercury is in these cases only in a very fine state of mechanical division.

BLUE PILL.

The account of an inquest on a person alleged to have died from the effects of *Blue pill*, will be found reported in the Medical Gazette, October 1843. It appears that the deceased, æt. 40, took some medicine prescribed for him by a practitioner. It consisted of six grains of blue pill and three of calomel. This was alleged to have produced salivation and a mercurial fever, of which the man died in about seven weeks. The salivation was probably owing to a remarkable idiosyncrasy; even a smaller dose than that here prescribed, has been known to cause fatal salivation. But from the evidence, it was not improbable that the deceased had taken some quack pills, which, had their composition been known, might have accounted for the severity of the symptoms. The jury returned a verdict of natural death, but called the remedy administered "an overdose of strong medicine." (Ph. Journ., Nov. 1843.) This compound can hardly be considered as an active poison. In doses of from five to ten grains it acts as a purgative. The only cases in which it has produced serious or fatal effects are those in which excessive salivation has followed its long-continued use in small doses.

Chemical analysis.—The odour and colour are sufficient to identify this compound. No globules of mercury can be seen if it be well prepared, but when rubbed on a clean surface of gold, it gives a white mercurial stain. By nitro-muriatic acid, the organic matter is decomposed and the mercury converted to corrosive sublimate. In this way it may be analyzed. Three grains of blue-pill contain one grain of mercury.

MERCURIAL OINTMENT. UNGUENTUM HYDRARGYRI.

This is a mixture of finely-divided mercury in a metallic state with lard and suet. It is of a blueish-grey colour, commonly used externally, and producing salivation and other mercurial symptoms by absorption. It is not often given internally, although its effects would be doubtless the same as those of blue pill. I lately had to examine many articles found in the possession of a woman charged with the crime of poisoning. There were several specimens of this ointment among them; but there was no proof that it had been administered as a poison. It would require a very large dose to produce any serious effects, and from its nauseous character it would probably be rejected by the stomach.

Chemical analysis.—Its colour and physical properties will serve to identify it. When well prepared no globules are visible to the eye, but it tarnishes with a mercurial stain a polished surface of gold. The fat may be separated by boiling in water: the particles of mercury subside, and any adhering portions of fat may be removed by digestion in ether or oil of turpentine. Two drachms of this ointment (Ung. Hyd. Fort.) contain one drachm of mercury. There is a more diluted preparation (Ung. Hyd. Mitius;) six drachms of this contain one drachm of mercury.

GREY POWDER. MERCURY WITH CHALK.

This a compound of carbonate of lime and finely-divided mercury. It is an innocent preparation, and acts as a purgative on children in doses of from three to five grains, and on adults in doses of from five to fifteen grains. It would require a very large dose to produce any injurious effects, and it is here enumerated among mercurial preparations because poisonous effects might be improperly ascribed to it. Its use long continued in small doses, produces the usual mercurial symptoms.

Chemical analysis.—It is of a greyish-blue colour, except when long exposed to light and heat, whereby it becomes lighter. When heated it yields a mercurial sublimate, and leaves carbonate of lime. Acetic acid dissolves the lime and leaves the mercury: nitric acid dissolves both. Globules of mercury are commonly visible in this powder by the aid of a lens. Eight grains of the powder contain three grains of mercury.

RED PRECIPITATE. RED OXIDE OF MERCURY.

This substance is commonly met with in crystalline scales of various shades of colour, from a dusky to a bright red, and forming an orange-coloured powder. It is poisonous, but instances of poisoning by it are very rare. The following case occurred at Guy's Hospital in 1833. A woman aged twenty-two, who had swallowed a quantity of red precipitate, was brought in labouring under the following symptoms. The surface was cold and clammy,—there was stupor approaching to narcotism,—frothy discharge from the mouth, and occasional vomiting:—the vomited matters contained some red powder which was proved to be red precipitate. There was considerable pain in the abdomen, increased by pressure; and there were cramps in the lower extremities. On the following day, the fauces and mouth became painful, and the woman complained of a coppery taste. The treatment consisted in the use of the stomach-pump, and the free administration of albumen and gluten. She left the hospital four days afterwards, still under the influence of mercury. The quantity of oxide here taken, was not ascertained.

Sobernheim reports a case, where a man, aged twenty-six, swallowed an ounce of red precipitate. He was speedily attacked with pain in the abdomen, nausea, purging, cramps and general weakness. The vomited matters consisted of masses of mucus, containing red precipitate. He continued to get worse, and died in less than forty-eight hours after taking the poison. On inspection, the mucous membrane was found eroded and inflamed in patches, small particles of the poison being imbedded in it. The duodenum was in a similar state, and there was a large quantity of red precipitate in the contents of the viscus, as well as in the stomach. (Op. cit. 250.) Niemann quotes a case in which six grains caused the death of a woman. (Taschenb. der Arzneiw. 452.) Notwithstanding the fatal result in these cases, red precipitate does not appear to be a very active irritant poison. *Thirty grains* of it have been taken and retained in the system, with comparative impunity. Some years ago a young woman swallowed this quantity, with a suicidal intention. Almost immediately afterwards, emetics of ipecacuanha and sulphate of zinc were administered, and the stomach-pump was used repeatedly; but none of the red powder was ejected. She suffered chiefly from pain in the abdomen and general weakness, but in the course of a few days she recovered. Devergie relates another instance of recovery in which a much larger dose had probably been taken. (Méd. Lég. ii. 705.)

A common opinion exists among the vulgar, that this compound is possessed of very active poisonous properties: hence it is sometimes administered with criminal design. At the Chester Lent Assizes, 1846, a woman was charged with attempting to poison a child by means of this substance. It was proved in evidence that the prisoner had given to a child, aged two months, about a teaspoonful of the nitric oxide. Vomiting of a reddish liquid speedily ensued; the greater part of the poison appears to have been thrown off the stomach, and the child recovered. The prisoner was convicted of the offence. The chemical evidence clearly established the nature of the compound; but a question arose whether it was to be considered a *deadly* poison or not, (see ante, page 16.) It has no claim to be so considered. (Med. Gaz. xxxvii. 874.)

The *Yellow wash*, made with a mixture of corrosive sublimate and lime water, owes its poisonous properties to precipitated red oxide. If the corrosive sublimate be in excess it may be an oxychloride. This lotion being in common use may easily give rise to accidents.

The nitric oxide has a local action as a stimulant, irritant, and escharotic. The ointment commonly used (Ung. Hyd. Nit. Oxydi) contains one-ninth part by weight of the oxide.

Chemical analysis.—Red precipitate is known,—1. By its being in red crystalline scales. 2. By its insolubility in water,—this, together with its great weight, renders it easy of separation from *organic liquids*. 3. It is readily dissolved by warm muriatic acid, forming a solution possessing all the properties of corrosive sublimate. 5. When heated in a small tube, it becomes black (reacquiring its red colour on cooling;) and while an abundant sublimate of metallic mercury is formed, oxygen gas is evolved. If the heat be continued, it should be entirely dissipated when pure,—a property by which it is known from most other red powders. In this experiment, a slight yellow sublimate is sometimes formed (subnitrate,) owing to the oxide retaining some traces of nitric acid.

IODIDES OF MERCURY.

The yellow and the scarlet iodides of mercury are irritant poisons which, judging from their effects on animals, are likely to act powerfully as irritants in the human subject. They have also a local action. They are quite insoluble in water, and both yield a mercurial sublimate when heated with carbonate of soda. The scarlet iodide is further known by its becoming yellow when heated, and again red on trituration.

CINNABAR. VERMILION. PERSULPHURET OF MERCURY.

The term *Cinnabar* is applied to a dark and heavy compound of sulphur and mercury, while *Vermilion* is the same substance reduced to a fine powder. It is well known as a red pigment, and is often employed in colouring confectionary, wafers, &c. I have not been able to find any instance of its having acted as a poison on man. Orfila believes that it is not poisonous. It has, however, proved fatal to animals in the proportion of from thirty to seventy grains, even when applied externally to a wound. Cinnabar is sometimes used for giving a red colour to ointments, *e. g.* the sulphur ointment. In such cases the quantity is very small, and can do no injury even if swallowed.

Dr. Sutro has published a short abstract of a case in which the *vapour* of *Vermilion* applied externally produced severe symptoms. A woman, by the advice of a quack, applied this vapour to a cancerous breast. She employed three drachms of vermilion, covering herself with a sheet so that the vapour should only reach the body externally. After three fumigations, she suffered from severe salivation and violent fever, which continued for four weeks. The right arm became œdematous. (Med. Times, Sept. 27, 1845, p. 17.)

Chemical analysis.—Vermilion is of a rich red colour, very heavy, and quite insoluble in water. When dropped into the hydrosulphuret of ammonia its colour remains unchanged; while red precipitate and red lead are turned of a dark brown colour, or even black. It is also known from red precipitate by its insolubility in muriatic acid. 1. When heated on platina it is entirely volatilized, the sulphur burning away. 2. Heated in a reduction-tube with carbonate of soda, a sublimate of metallic mercury is obtained with a residue of sulphuret of sodium, in which sulphur may be easily proved to exist by the usual tests, *e. g.* by placing a portion of the residue on a glazed card and adding a drop of water. (See p. 174, ante.)

BICYANIDE OF MERCURY.

This is a substance which is but very little known, except to chemists, yet it is an active poison, and has caused death in one instance. In April 1823, a person who had swallowed twenty grains of this compound (thirteen decigrammes,) was immediately seized with all the symptoms of poisoning by corrosive sublimate, and died in nine days. There was continued vomiting with excessive salivation, ulceration of the mouth and fauces, suppression of urine, purging, and, lastly, convulsions of the extremities. On inspection, the mucous membrane of the stomach and intestinal canal, was extensively inflamed. (Orfila, i. 583.) Dr. Christison quotes a case in which ten grains destroyed life within the same period of time. (On Poisons, 427.) As a poison, the bicyanide is probably not much inferior in activity to the bichloride of mercury.

Chemical analysis.—This is a white salt commonly crystallized in quadrangular prisms. It is very soluble in water, especially at the boiling point. The solution has no odour of prussic acid until an acid is added. Its solution is neutral, and is not precipitated by caustic potash. 1. If the powdered crystals be heated in a small tube, cyanogen gas is evolved and may be burnt at the mouth, the flame being of a rich rose-red colour, with a blue halo:—metallic mercury is at the same time sublimed. This experiment is alone sufficient to identify the poison. The solution of bicyanide is not unlikely to be mistaken for that of corrosive sublimate. It readily deposits mercury on copper and gold by the galvanic test, and it is precipitated black by hydrosulphuret of ammonia and sulphuretted hydrogen gas. It is known, 1. By potash producing in it no precipitate; 2. By prussic acid being evolved on boiling a portion of it with muriatic acid, corrosive sublimate being produced in the liquid; 3. By a scarlet precipitate being formed on adding to the solution, iodide of potassium and muriatic acid.

TURBITH MINERAL. SUBSULPHATE OF MERCURY.

Fatal cases of poisoning by this compound are by no means common. It is undoubtedly, although very insoluble, a strong irritant poison, and is capable of causing death in a comparatively small dose. A well-marked instance of its fatal operation was communicated to the Pathological Society by Mr. Ward in March 1847. A boy, æt. 16, swallowed *one drachm* of this preparation on the night of February 19th. It produced a burning sensation in the mouth and throat, and vomiting in ten minutes. In about an hour there was paleness with anxiety of countenance, coldness of surface, constant sickness, sense of heat and constriction in the throat, and burning pain in the stomach with cramps. The irritability of the stomach continued in spite of treatment, and after two days, there was salivation with mercurial fœtor. The gums acquired a deep blueish tint and began to ulcerate. The patient died in about a week after he had taken the poison, without convulsions, and without suffering at any period from symptoms of cerebral disturbance. The principal post-mortem appearances were,—inflammation of the œsophagus; its mucous membrane at the lower part peeling off;—the inner surface of the stomach near the cardia and pylorous, was covered with petechial spots;—the small intestines were contracted, the inner coat reddened and petechial spots were found, but chiefly in the large intestines. The parotid and submaxillary glands were swollen. Mercury was detected in the intestines (see Med. Gaz. xxxix. 474.) From this account, it will be perceived that turbith mineral has an action somewhat similar to corrosive sublimate, although it is probably much less active.

Chemical analysis.—Turbith mineral is a heavy powder of a yellow colour, becoming of a dark olive by exposure to light. It is scarcely soluble in water, but has a strong metallic taste. When heated in a tube, with or without carbonate of soda, it yields metallic mercury. It is best analyzed by boiling it in potash, in which case sulphate of potash and peroxide of mercury result—the acid and the base are then easily determined.

NITRATES OF MERCURY.

These are corrosive poisons which are used for several purposes in the arts. They are solid white salts, easily dissolved by water, especially if there be a little excess of acid present. The acid pernitrate has already caused death in an interesting case reported by Mr. Bigsley in the Medical Gazette (vi. 329.) A butcher's boy dissolved some mercury in strong nitric acid, and swallowed about a teaspoonful of the solution. Soon afterwards he suffered the most excruciating pain in the pharynx, œsophagus and stomach:—there was great anxiety, with cold skin, small pulse, colic and purging. He became gradually weaker, and died in about two hours and a half. On inspection the fauces, œsophagus and stomach, were found corroded and inflamed. Although he survived so short a time, the mucous membrane of the stomach was of a deep red colour.

The solution of acid pernitrate has been used as a cautery, and when diluted, as a lotion in diseases of the skin. Under these circumstances, it has been known to cause severe salivation, sloughing, and suppression of urine. (See p. 320.) A medical practitioner in France, was lately charged with malapraxis, in having improperly prescribed this solution as a wash for the itch. The skin of the body where the lotion had been employed, was discoloured and partly corroded; there was also pain in the throat, difficulty of swallowing, inflamed gums and salivation. The parties recovered from these effects in the course of a short time, and the charge was dismissed on the ground, that the lotion had been improperly used by the complainants; they had used it in a concentrated state, instead of diluting it with water, as ordered by their medical attendant. (Ann. d'Hyg. Juillet 1842.) A case of death from this compound has been recently reported (Journal de Chimie Méd. 1846, p. 734.) There can be no doubt, that this poison is quite as formidable, both as a corrosive and irritant, as corrosive sublimate itself. The diluted protochloride of tin, if timely applied, will counteract the local effects of these corrosive compounds of mercury. The nitrate of mercury is used in the form of ointment (UNG. HYDR. NITRATIS) as a local application. A dose of this might exert an irritant action. About ten parts of the ointment contain three of the nitrate.

CHEMICAL ANALYSIS.—In the solid state, the crystals of acid *Pernitrate*, when heated in a tube, yield nitrous acid vapour,—peroxide of mercury, and a ring of metallic mercury,—when heated with carbonate of soda, metallic mercury is easily obtained. In *solution*, it is commonly met with in the state of a highly acid liquid, and nitric acid may be obtained by distillation, a result which does not occur with a mere solution of the crystals in water. The solution possesses all the properties of corrosive sublimate, so far as the tests for mercury are concerned; but it gives no precipitate with nitrate of silver. On adding carbonate of potash and filtering, nitrate of potash is easily detected in the filtered liquid, and thus the acid is identified. On evaporating to dryness, and obtaining the solid crystals, they may be easily known from those of corrosive sublimate, by the simple application of heat. The *Protonitrate* is known in solution, both from pernitrate and corrosive sublimate, in its being precipitated black by potash, and white by muriatic acid. This acid does not precipitate the solutions of either of the salts above mentioned.

ACETATE OF MERCURY.

The ACETATE is another soluble salt of mercury, but nothing is known respecting it as a poison.

PROPORTION OF MERCURY IN PHARMACEUTICAL PREPARATIONS.—It may be proper to insert in this place the proportion in which mercury enters into various medicinal compounds belonging to the London Pharmacopœia, some of which, by causing death, have given rise to important medico-legal inquiries. Mercury with chalk (HYDRARGYRUM CUM CRETA) contains three grains of mercury in eight grains of powder. Blue pill (PILULÆ HYDRARGYRI) contains one grain of mercury in three grains. Compound calomel pill (PILL. HYD. CHLOR. COMP.) contains one grain of calomel in five grains. Iodide of mercury pill (PILL. HYD. IOD.) contains one grain of the iodide in five grains. Solution of corrosive sublimate (LIQUOR HYDRARGYRI BICHLORIDI) contains one grain of corrosive sublimate in two ounces of solution. The dose is from half a fluid-drachm to two fluid drachms. For *external applications.*—Compound mercurial liniment (LIN. HYD. COMP.) contains about ten grains of mercury in one drachm. Strong mercurial ointment (UNG. HYD. FORT.) contains one drachm in two drachms; and the mild mercurial ointment (UNG. HYD. MITIUS) contains one drachm in six drachms. The red precipitate ointment (UNG. HYD. NIT. OXYDI) contains one-ninth of its weight of red nitric oxide of mercury. The yellow or golden ointment (UNG. HYD. NITRATIS) contains, in ten parts, about three parts of the nitrate.

CHAPTER XXVII.

ON POISONING BY LEAD—ACTION OF LEAD-SHOT—WHAT SALTS OF LEAD ARE POISONOUS? —SUGAR OF LEAD—SYMPTOMS—CHRONIC POISONING BY SUGAR OF LEAD—POST-MORTEM APPEARANCES—TREATMENT—QUANTITY REQUIRED TO DESTROY LIFE—RECOVERY FROM LARGE DOSES—GOULARD'S EXTRACT—CHEMICAL ANALYSIS—DELICACY OF THE TESTS—OBJECTIONS TO THEIR USE—LEAD IN ORGANIC MIXTURES—IN THE TISSUES. NITRATE OF LEAD—CHLORIDE—OXYCHLORIDE—CARBONATE OR WHITE LEAD—CASES OF POISONING—PAINTER'S COLIC—ABSORPTION OF LEAD—ACCIDENTS FROM THE CARBONATE OF LEAD—POISONING OF WATER BY LEAD—SULPHATE OF LEAD—CHROMATE —IODIDE—OXIDES—LITHARGE AND RED LEAD—ACCIDENTS FROM THE GLAZING OF POTTERY—LIQUIDS POISONED BY SHOT—EFFECTS OF EXTERNAL APPLICATION—HAIR-DYES.

General remarks.—The metal Lead is not commonly regarded as poisonous, but it is readily susceptible, by exposure to air and moisture, of being converted to a poisonous salt,—the carbonate of lead. Many of its compounds are also much used in the arts, and have, on several occasions, given rise to serious accidents. Dr. Bryce has reported an interesting case, from which it would seem that lead is capable of exerting a poisonous action even in the *metallic* state. A man, aged twenty-three, swallowed three ounces of small shot (No. 4,) in three days. On the third day there was great anxiety and depression, with sunken features, coldness of surface, dizziness, and numbness in the arms and legs. He continued getting worse in spite of treatment; his bowels were obstinately torpid, and there was increased numbness in the arms, and dizziness. Purgatives were exhibited; the alvine discharges were examined, but only one pellet was found, so that if he passed the shot at all, it must have happened in the three days before he was seen by Dr. Bryce. This man perfectly recovered in a fortnight. (Lancet, Dec. 31, 1842.) Shot are known to be formed principally of lead, with some small portion of arsenic in the state of arsenuret or arseniate of lead. The quantity of arsenic is probably less than the 200th part of the weight of the shot; but the symptoms here appeared to show, that the effects were due to lead, and not to arsenic; the metal was probably oxidized and converted to an organic compound of lead, by the acid-mucous secretions of the stomach. This case justifies the opinion that metallic lead cannot be strictly regarded as inert, or, if inert as a metal, it is susceptible of becoming speedily transformed to a poisonous salt within the body. The only compounds of lead which have been found to produce poisonous effects upon the system, are the acetate, subacetate, chloride, carbonate, and the oxide of the metal combined either with vegetable acid or fatty substances. Dr. A. T. Thomson has expressed an opinion that the carbonate of lead is the only poisonous salt of this metal: and that, if any other salt in small doses become poisonous, it is merely by its conversion to carbonate within the body; but as he admits that the acetate and subacetate may act as irritant poisons in large doses, and no toxicologist maintains that they are poisons when taken in small quantity, the difference of opinion appears to be more verbal than real. (See Med. Gaz. x. 689.) So far as observations on man have yet extended, the carbonate has no more action than the common acetate, (p. 350.) Dr. C. G. Mitscherlich has, however, proved, that the acetate of lead is a poisonous salt; and that when mixed with acetic acid, it is more energetic than when given in the neutral state. This fact clearly shows that the poisonous effects cannot solely depend on the assumed conversion of the salt to the state of carbonate. (Brit. and For. Med. Rev. No. vii. 208.) Besides, it is not easy to perceive how the nitrate and chloride should become converted to carbonate within the body. Dr. Thomson has kindly furnished me with the following statement with reference to his views. When the salts of lead operate as poisons in moderate doses, he has no doubt that they are converted into the carbonate of lead in the stomach. He gave with impunity ten or twelve grains of acetate of lead every sixth hour in cases of severe active hæmorrhages, when it was washed down with distilled vinegar, whereas without the vinegar, colica pictonum would have supervened. He has also known instances in which this disease as well as paraplegia, were induced by acetate of lead, when washed down with soda powders in a state of effervescence and no vinegar was given.

[Lead is capable of producing poisonous effects in the state of vapor, as is shown by the fact, that casters in this metal are constantly liable to attacks of colica pictonum.—G.]

SUGAR OF LEAD. ACETATE.

This is more frequently taken as a poison than any of the other salts, although cases of acute poisoning by lead in any form, are very uncommon. In the Coroners' report for 1837–8, there is not a single instance. The substance is commonly met with in solid heavy crystalline masses, white or of a brownish-white colour; it much resembles loaf-sugar in appearance, and has often been mistaken for it. It has also a sweet taste, which is succeeded by an astringent or metallic taste. It is very soluble in water. Four parts of water at 60° will dissolve one part; and it is much more soluble at a boiling temperature. It is soluble in alcohol. I am informed by a respectable druggist that sugar of lead is retailed to the public at the rate of three-halfpence an ounce, and that for quantities less than this, one penny is charged.

SYMPTOMS.

Acetate of lead is by no means an active poison, although it is popularly considered to possess a very virulent action. In medical practice, it has often been given in considerable doses without any serious effects resulting. Dr. Christison states that he has often given it in divided doses to the amount of eighteen grains daily for eight or ten days without remarking any unpleasant symptom whatever, except, once or twice, slight colic. (Op. cit. 555.) When, however, the quantity taken has been from one to two ounces, the following symptoms have been observed. A burning pricking sensation in the throat, with dryness and thirst:—vomiting supervenes; there is uneasiness in the epigastrium, which is sometimes followed by violent colic. The abdomen is tense, and the parietes have been occasionally drawn in. The pain is relieved by pressure, and has intermissions. There is in general constipation of the bowels. If any fæces be passed, they are commonly of a very dark colour, indicative of the conversion of lead to sulphuret. The skin is cold, and there is great prostration of strength. When the case is protracted, the patient has been observed to suffer from cramp in the calves of the legs, pain in the insides of the thighs, numbness and sometimes paralysis of the extremities. The affection of the nervous system is otherwise indicated by giddiness, torpor, and even coma. A well-marked blue line has been observed round the margin of the gums, where they join the teeth.

The following case presents the ordinary train of symptoms which are observed in acute poisoning by acetate of lead. It adds another to the numerous instances already recorded of recovery from a very large dose of this salt. M. A——, aged 41, was admitted into Guy's Hospital, May 1846, labouring under symptoms of poisoning by sugar of lead. It was ascertained that two hours previously, she had swallowed about one ounce and a half of sugar of lead, or half a tea-cupful dissolved in some water. She felt ill almost directly; had a nauseous metallic taste in her mouth, with a burning heat in the mouth, throat, and stomach. She took some more water to wash down the taste. This made her vomit, and thus she was detected by her friends. Her mouth became very dry; had great pain at the pit of the stomach, and excessive vomiting.

On admission, *i. e.* about two hours after the poison had been taken, she felt sleepy and stupid, alternately perspiring and shivering. Complained of violent twisting pain in the abdomen, but this was relieved by pressure; with this, there was a sensation of sickness. She felt exceedingly weak and languid; complained of a feeling of cramp in the thighs, and numbness all over the body, with giddiness. The gums felt to the patient to be in lumps, and there was apparently a blue line on the edge: they were very tender. The saliva seemed rather in excess; and the breath was foul. Magnesia mixture and sulphate of zinc were given to encourage the vomiting; followed by some castor-oil, which acted two hours afterwards. The pulse was hurried, and the tongue coated; countenance anxious and excited; skin dry, cold and hot alternately. The urine was passed very freely. Was menstruating at the time, and this stopped the discharge. Breathing impeded from pain in the bowels. The next day there were pains all over the body, with numbness and sickness. On the third day she was very sleepy, but in less pain. For several days the abdomen was excessively painful on the slightest pressure. She left the hospital in five days.

Even when the patient recovers from the first symptoms, the secondary effects often last for a considerable time. In two cases which occurred to Mr. Gorringe, two girls swallowed an ounce of the acetate of lead by mistake. Soon afterwards they felt a burning pain in the mouth, throat, and stomach, and in a quarter of an hour they vomited freely: in half an hour there was severe pain in the bowels, with diarrhœa. Under treatment, recovery took place. (Prov. Med. Journ. April 1846.) Although nearly a year had elapsed, they both suffered from severe pain in the epigastrium, which was tender on pressure. Nothing could be retained on the stomach: and there was a choking sensation in the throat, with other constitutional symptoms. Paralysis and other symptoms of nervous disorder are, however, by no means necessary consequences. A girl who had swallowed sixty grains of acetate of lead, and suffered severely from the primary symptoms, recovered and left the hospital in about three weeks without any paralysis or other disorder affecting the muscular system. (Lancet, April 4, 1846, p. 384.) This lead-palsy would appear to be a more common consequence of small doses frequently repeated.

The symptoms are sometimes very slow in appearing. The following case occurred to Dr. Hviding. A girl swallowed about three drachms of the acetate of lead in breth. It was not until *two hours* afterwards that she began to experience sharp colicky pains in the abdomen, followed by vomiting. No medical treatment was employed for three days: and the only marked symptom then, was obstinate constipation. Doses of castor-oil were prescribed, and the girl recovered. (Journal de Chimie, 1845, 256.)

An inexplicable form of poisoning by this compound has lately come to light in Belgium. A druggist of Maestricht, well-acquainted with chemistry, was attacked with violent colic in common with other members of his family after a meal. He examined the bread, water and other articles of food, and he found that the *butter* contained a very large quantity of acetate of lead. The object of this nefarious adulteration was not at all apparent. (Journal de Chimie Médicale, 1845, 673.)

Chronic poisoning.—A very interesting case of this form of poisoning by the acetate has been communicated to the Pharmaceutical Journal, by Dr. Letheby. (December 1845, p. 259.) A child æt. 6, took, in a quack-medicine, 1-5th of a grain of acetate of lead two or three times a day for nearly nine weeks. It was then found to be labouring under symptoms of poisoning by lead, and two days afterwards the child died. The first effects of taking the medicine, were that the child fell away, and complained of colicky pains: the bowels were constipated, stools black and offensive, and there was fœtor of the breath. Latterly the child was very drowsy and the limbs were paralyzed. Upon the day of its death it became convulsed, and shortly before death it fell into a state of coma.

POST-MORTEM APPEARANCES.

On an inspection of the body, in the case just related, the skin was of a dingy yellow, and the gums were of a deep blue colour. The lungs were slightly congested, and there was an effusion of serum in the pleuræ. The blood was black and liquid. The stomach and intestines were pale and nearly empty,—the former contained half an ounce of a thick brownish fluid, in which lead was detected; the latter were contracted in some places and dilated in others, and they presented several points of intus-susception. The large intestines were in a similar condition. The bronchial and mesenteric glands were enlarged. The bladder and ureters were full of urine—the rest of the viscera healthy. Lead was freely detected in the brain, muscles, liver, intestines, the blood, and in the serum found effused in the ventricles; but none was discovered in the bile or urine. It was calculated that the child had taken during the nine weeks from 26·6 to 33·3 grains of acetate of lead. The fact that not more than one-fifth of a grain had been taken in one day, and that two days had intervened before death without any portion having been administered, shows how very slowly this poison is eliminated. (For additional remarks on chronic poisoning by lead, see COLICA PICTONUM, page 360, post.)

In one *acute* case related by Dr. Kerchhoffs, the mucous membrane of the stomach was found abraded in several places, especially near the pylorus; and most of the abdominal viscera were in a state of high inflammation. A trial for murder by this substance took place at the Central Criminal Court, in November 1844, *Reg.* v. *Edwards*, but the details are so imperfectly reported, as to throw no light upon the subject. The stomach and intestines are stated to have been found inflamed, and there were dark spots on the former. In animals, according to Dr. Mitscherlich, when the dose is large, the mucous coat of the stomach is attacked and corroded; this change appears to be purely chemical, and takes place in all the organs of the body with which the salt of lead comes in contact. If given in a small dose, it is decomposed by the gastric secretions, and exerts no corrosive power on the mucous membrane. When the acetate of lead was given in a state of albuminate dissolved by acetic acid, death took place with great rapidity; but on inspection, the stomach was not found to be corroded. This corrosive action belongs to the neutral salt, and is not manifested when the dose is small, or when the poison is combined with an acid.

QUANTITY REQUIRED TO DESTROY LIFE.

Nothing is accurately known concerning the *fatal dose* of sugar of lead. The facts already detailed show that it may be taken in comparatively large quantities, without producing serious effects. Thirty and forty grains have been given daily, in divided doses, without injury. The following additional cases, in some of which recovery took place under very disadvantageous circumstances, prove that the acetate of lead is far from being a virulent poison. Dr. Iliff met with an instance where an ounce was swallowed in solution. The symptoms were pains in the abdomen resembling cholic, vomiting, rigidity, and numbness. It was three hours before any remedies were used, and five hours before the stomach-pump was employed; but the person recovered! In the second case an ounce was swallowed: sulphate of magnesia was freely exhibited, and the stomach-pump was used. On the following morning there was slight excoriation of the gums, which were white, with a sensation of heat in the throat: the bowels were relaxed, probably from the effect of the medicine. The day following, there were pains in the calves of the legs and thighs, with restlessness and thirst. In a week the woman perfectly recovered. In October 1835, a girl, aged nineteen, dissolved about an ounce of acetate of lead in a cupful of water, and swallowed it. In a quarter of an hour violent vomiting came on, and she was taken to the North London Hospital. Sulphate of magnesia and diluted sulphuric acid were given to her; there was slight pain in the abdomen, weight in the head, dimness of sight, with pains shooting through the eyeballs. The abdomen was tender on pressure for several days; but in five days the patient was discharged cured. The fourth case occurred in Paris, in 1840. A girl swallowed *an ounce* of the acetate of lead: the usual symptoms followed, and sulphate of soda was administered. She recovered. (See p. 348.) In a case recently reported by Dr. Evans, a woman recovered, after having swallowed half an ounce of the acetate by mistake, under the free use of aromatic sulphuric acid. (Amer. Journ. Med. Sciences, February 1847, 259.)

The dose of this poison required to destroy life became a question in the case of *Reg.* v. *Hume* (Chelmsford Summer Ass., 1847.) The prisoner was charged with an attempt to murder her husband, by endeavouring to administer to him "a large quantity of a certain *deadly poison* (on the absurd use of these terms see p. 17, ante,) called sugar of lead!" According to the evidence, she made two large boluses with flour and water; and the quantity of sugar of lead contained in them, was equal to *twenty-six grains and a half.* The prosecutor refused to take the pills. The prisoner admitted that she intended to administer them to him, but not with the design of poisoning him. The prosecutor was in a bad state of health: and it was alleged that, under these circumstances, twenty-six grains and a half of the sugar of lead would certainly have caused his death. The prisoner was convicted, and sentenced to transportation for life. Admitting the report of the evidence to be correct, it is impossible to reconcile the facts known concerning the effects of the acetate of lead, with the strong medical statement here made. So far as I have been able to ascertain, there is not a single instance recorded, in which even sixty grains have destroyed life. Van Swieten gave it to the amount of one drachm daily for ten days before it caused any material symptom. (See Christison, Op. cit. 555.) In another case, violent symptoms were produced by this dose, but the individual easily recovered from the effects. The observations and experiments of Orfila also prove that the vulgar belief of sugar of lead being an active poison, is erroneous. Even admitting that the prosecutor was in a bad state of health, the assertion that less than half a drachm of sugar of lead would have caused his death, is quite unsupported by any of the facts hitherto ascertained respecting the effects of this compound.

TREATMENT.

This consists in the free exhibition of solutions of the alkaline sulphates, either of soda or magnesia. The carbonates should be avoided, as the carbonate of lead is poisonous; while the sulphate is either inert, or possesses but very little activity. Purified animal charcoal has been recommended as an antidote, in consequence of the property which it possesses, to a certain extent, of separating oxide of lead from its saline combinations. My experiments on this subject lead me to infer, that it is as inefficacious with respect to the salts of lead, as with respect to arsenic and corrosive sublimate (ante, p. 78.) One grain of acetate of lead was dissolved in an ounce of water; and while the solution was still warm, it was shaken with thirty grains of animal charcoal. The oxide of lead was found to have been immediately removed; and the filtered liquid was acid, showing that acetic acid remained. One grain of the acetate was then shaken with thirty grains of animal charcoal and five drachms of cold water. The oxide of lead was entirely removed in ten minutes. The same effect was observed when the charcoal was in the proportion of twenty parts to one of the acetate; but when reduced to twelve parts to one, the salt of lead was retained in solution. Five grains of the acetate were mixed with twenty grains of animal charcoal and a similar quantity of water, and the mixture was frequently shaken for twenty-four hours. The hydrosulphuret of ammonia, iodide of potassium, and other tests, showed that lead was still abundantly present in the filtered liquid. The power of charcoal to separate oxide of lead is therefore very limited. If given in a proportion of less than twenty or thirty times the weight of the poison, it would probably have no counteracting effect. Vegetable charcoal, in the proportion of sixty parts to one of acetate of lead, entirely removed the oxide of lead, but when the proportion was reduced to *thirty* parts, lead was still held in solution.. This effect of charcoal is however important, in showing that this substance cannot be safely employed for decolorizing liquids containing lead. The reader will find some remarks on this subject by M. Chevalier. (Ann. d'Hyg., 1845, i. 135.)

An emetic of sulphate of zinc should be given, if vomiting does not already exist. The stomach-pump may be occasionally employed with benefit. It is well-known that albumen precipitates the oxide of lead when added in large quantity; and Mitscherlich has found that casein, the albuminous principle of milk, is a very effectual precipitant of the oxide of lead. Therefore it would be advisable to administer, in cases of poisoning by the soluble salts of lead,—milk or albumen in large quantity. The compounds thus formed, as in the case of corrosive sublimate, may not be absolutely inert; but they are far less active than the acetate itself, and tend to prevent the action of the poison as a corrosive on the stomach. Five cases have been mentioned where individuals recovered partly through treatment, after having swallowed one ounce of the acetate of lead.

CHEMICAL ANALYSIS.

Acetate of lead as a solid.—1. If a portion of the powder be heated in a small reduction-tube it melts, then becomes solid; again melts, acquiring a dark colour, and gives off vapours of acetic acid; a black mass is left in the tube, consisting of carbon and reduced metallic lead. There is no sublimate formed. 2. It is very soluble in water, even when cold; common water is turned milky by it, chiefly from the presence of carbonic acid and sulphates. 3. A small portion of the powder dropped into a saucer, containing a solution of iodide of potassium, acquires a fine yellow colour. 4. When dropped into caustic potash, it remains white;—5. Into hydrosulphuret of ammonia, it is turned black, in which respect it resembles the white salts of some other metals. 6. When the powder is boiled in a tube with diluted sulphuric acid, acetic acid, known by its odour and volatility, escapes. All these properties taken together, prove that the salt is acetate of lead.

Acetate of lead in solution.—If acetate of lead be presented in a state of solution, or if the solid salt be dissolved in water for the purpose of making further examination, we should note the following points. 1. A small quantity, slowly evaporated on a slip of glass, will give white and opaque prismatic crystals, which are turned yellow by iodide of potassium, and black by hydro-sulphuret of ammonia. The solution is said to be neutral; but I have found the common acetate of lead to have at the same time both an acid and an alkaline reaction, i. e. reddening litmus-paper, and turning rose-paper green, a circumstance which might create some embarrassment in an analysis. 2. *Caustic potash*, added to the solution much diluted with water, throws down a white precipitate, which is easily soluble in an excess of the alkali. 3. *Diluted sulphuric acid* produces an abundant white precipitate, insoluble in nitric acid, but soluble in muriatic acid and in a large excess of caustic potash. 4. It is precipitated of a bright yellow colour by the *Iodide of potassium;* the yellow iodide of lead is soluble in caustic potash, forming a colourless solution. It is also dissolved by concentrated muriatic acid. 5. *Hydrosulphuret of ammonia* or sulphuretted hydrogen gas, produces a deep black precipitate, even when less than the 100,000th part of the salt is dissolved. 6. Place a few drops of the solution on clean platina-foil,—acidulate it with acetic acid, then apply through the solution, to the surface of the platina, a thin polished slip of zinc:—bright crystals of metallic lead are instantly deposited on the zinc: in this way a very small quantity of lead may be detected. 7. If a slip of zinc-foil with a little acetic acid be introduced into the solution, there speedily takes place a deposit of metallic lead on the surface of the zinc. 8. If bibulous paper be repeatedly saturated with this or any soluble salt of lead, dried and burnt, metallic lead is reproduced in minute globules, which may be collected and examined.

Delicacy of the tests.—The following results were obtained by a series of experiments on the action of three of the tests. The 1-440th part of a grain of acetate of lead, dissolved in the smallest possible quantity of distilled water, gave readily the reactions indicated with diluted sulphuric acid, iodide of potassium, and hydrosulphuret of ammonia;—the change of colour produced by the two last tests being exceedingly well marked. When 1-15th of a grain of acetate was diffused in an ounce of water, sulphuric acid produced no effect:—a precipitate began to be formed only when the quantity of acetate amounted to 1-5th of a grain. The effect of iodide of potassium is entirely destroyed if the salt of lead be much diluted. This test wholly failed to detect half a grain of acetate in twelve ounces of water, and it was found that a small quantity of acetate (1-15th grain,) which gave a rich yellow precipitate when dissolved in a few drops of water, was not perceptibly affected by the test when six drachms of water were previously added. The hydrosulphuret of ammonia was much less affected than the other tests by the diluted state of the salt of lead; 1-220th part of a grain of acetate, diffused in twelve ounces of water, acquired a pale brown tint on the addition of the test: this became deeper with the 150th, and very decided with the 30th part of a grain: although the poison was here diffused through 158,400 times its weight of water.

Objections to the tests.—It must be understood that the effects of the tests should be always taken together, as the objection to any one is thus counteracted by the application of the others. With regard to potash, this gives a similar precipitate with the salts of barytes, strontia, lime, and magnesia; but none of these precipitates are soluble in an excess of the alkali. Potash also precipitates alum and sulphate of zinc; and these precipitates, like that from lead, are soluble in an excess of caustic alkali; but the solutions of alum and zinc are not precipitated by sulphuric acid. All the precipitates of lead which are redissolved by caustic potash, are thrown down black by adding hydrosulphuret of ammonia to the respective alkaline solutions. This enables the analyst to apply two or three tests to one portion of suspected liquid. Sulphuric acid gives a white precipitate with some other compounds; but these are known from sulphate of lead, by their insolubility in caustic potash, while the sulphate of lead is known from them by its solubility in concentrated muriatic acid. The action of iodide of potassium is peculiar. There are several objections to the fourth test; for the salts of mercury, silver, copper, cobalt, nickel, bismuth, and the protoxides of iron and tin, are precipitated by hydrosulphuret of ammonia, either of a black or dark brown colour (p. 131.) Some of the solutions of these salts are known by certain specific properties,—those of copper, cobalt, and nickel, are coloured;—the acid solution of bismuth is decomposed and precipitated by water, while the salts of these metals do not give results similar to those of lead with the other tests, nor can lead be extracted from them by the galvanic or any other process.

Lead as an impurity in liquids.—The analyst should be aware, that lead may be contained in many alkaline or saline liquids when they have been long kept in flint-glass bottles. Thus, solutions of potash and soda, as well as of their respective carbonates, become so strongly impregnated with lead, after having been kept a few weeks in flint-glass, as to be turned of a dark brown colour, or even black, on the addition of hydrosulphuret of ammonia. I have found so much lead in a solution of caustic soda thus kept, that it was precipitated by sulphuric acid. That the lead was derived from this source, was clearly proved by the fact, that the solutions, before being placed in flint-glass were not affected by the hydrosulphuret. It is said that the alkaline chlorides and phosphates, as well as the mineral acids, will also dissolve the oxide of lead under these circumstances; but I have not found this to be the case. These facts should be borne in mind when liquids are examined which are suspected to have been intentionally poisoned. The quantity of oxide of lead thus dissolved, is not sufficient to produce mischief; but its presence might lead to an erroneous opinion and embarrass the analysis (p. 118, ante.)

Lead in organic mixtures.—The acetate of lead is precipitated by many organic principles, especially by albumen and tannin. Thus we may have to analyse either an organic liquid containing lead, or a solid precipitate consisting of mucus or mucous membrane, intimately united to the oxide of lead. The liquid must be filtered and examined by a trial test, *i. e.* either by adding to a portion, sulphuric acid, or by exposing bibulous paper dipped into the suspected liquid, to a free current of sulphuretted hydrogen gas. If the paper be not stained brown, there is no perceptible quantity of lead dissolved;—if it be stained brown, we dilute the liquid if necessary in order to destroy its viscidity, and pass into it a current of sulphuretted hydrogen until all action has ceased. The black sulphuret of lead should be collected on a filter, washed and dried, then boiled for a quarter of an hour in a mixture of one part of nitric acid, diluted with four parts of water. This has the effect of transforming it, at least in part, to nitrate of lead soluble in water. This liquid, when filtered, may be cautiously neutralized by potash or ammonia (free from lead,) and the tests added. If the quantity be too small for the application of all the tests, we may add sulphuric acid; if a white precipitate be formed, soluble in potash, and this solution be again turned black by hydrosulphuret of ammonia, this is sufficient evidence of the presence of lead. Should there be no lead dissolved, we must decompose the solid and insoluble matters in nitric acid slightly diluted, at a boiling temperature, filter, and test the filtered liquid, previously neutralized; or we may evaporate to dryness, carbonize by nitric acid, and redissolve the residue in water for testing.

QUANTITATIVE ANALYSIS.

This may be most conveniently effected with respect to any of the soluble salts of lead, by passing into the solution, a current of sulphuretted hydrogen gas, until the filtered liquid gives no longer any proof of the presence of lead. The precipitate should be well washed, dried, and weighed. Every 100 parts of sulphuret are equal to 158·3 of crystallized acetate: 138·3 of crystallized nitrate: 116·6 of chloride, and 111·6 of carbonate of lead. If the salt of lead be insoluble in water and acids, it is merely necessary to diffuse it through water in an impalpable mixture, before passing into it the current of sulphuretted hydrogen gas.

Detection of lead in the tissues.—If no result be obtained by either of the above-described processes, it will be necessary to cut the stomach, or other suspected solids, to pieces; and after having dried them thoroughly, to incinerate them with four parts of black flux in a crucible. If lead be present in large quantity, it will be found at the bottom of the crucible in the form of a metallic button, or in heavy grains, and the carbonaceous matters may be separated by washing the residue in water. The nature of the acid united to the lead, cannot of course be determined in such a case; but this would be unimportant to the inquiry, as all the salts of lead, excepting perhaps the sulphate, are poisonous. In poisoning by the subacetate, traces of lead have been found in the stomach three days after death; and there is no doubt that it might be detected after many months had elapsed.

In November 1843, an interesting trial took place at the Assizes of the Puy de Dôme, in France, involving the rare question whether the death of a person had or had not been caused by the criminal administration of a salt of lead. The deceased died under suspicious circumstances:—on an inspection of the body nothing was observed to indicate the action of an irritant poison, while the stomach was ulcerated, and in an otherwise diseased condition. No salt of lead was found in the contents, but traces of the metal were discovered on incinerating the viscera. The question then arose, whether the metal thus found was a natural constituent of the body, or the result of a portion which had been swallowed, and had acted as a poison. The medical opinions were conflicting. Orfila thought it was very probable, if not certain, that the deceased had died from the effects of lead. There was so much doubt about the case, that in an English Court of Law, it would probably have been speedily dismissed for want of clear medical evidence of the cause of death. There was an entire failure of proof. (Annales d'Hygiène, Janvier 1844.)

In the following case, the fact of lead-poisoning was made evident only by the discovery of lead in the tissues, suspicion having been excited by the nature of the symptoms under which the female was labouring when admitted into the hospital. It indicates also the plan to be pursued when the quantity of lead is very small. A female, aged 27, was admitted into Guy's Hospital in April 1846. For some months she had been leading a most irregular life, and it was supposed, by the people with whom she had been living, that she had taken something to produce abortion. About ten weeks before her admission, she was suddenly attacked by vomiting and cold shivering, with severe pain in the knees. This continued for some days, after which there were dull, aching pains in the stomach and back. Three weeks previously she had been treated for chronic gastritis, with some benefit. On a relapse she was sent to the hospital. Her bowels, during the whole ten weeks, had only been opened under the use of medicine. The third day after her admission, a distinct blue line was noticed on both gums, and it was found that she could not extend either wrist to a full extent. The day following, the paralysis of the extensors of the wrist had become much more marked, and the hands trembled very much. She became insensible, and died a week after her admission. It could not be ascertained from her statements, that she had taken any poison. On inspection, a large quantity of clear serum was found beneath the arachnoid; the brain was pale and bloodless. The muscular coat of the stomach was hypertrophied, especially towards the pylorus, where it constricted the opening; the mucous membrane of the stomach was thick and rugose. The large intestines were irregularly contracted and distended; there were spots of congestion scattered on the mucous coat; the salivary glands were enlarged.

As there was a suspicion, from the state of the gums and other symptoms, that the deceased had, at some previous time, taken lead as a poison, the liver was dried and incinerated, and the ash thus obtained was digested in water, containing one-eighth part of strong nitric acid. The acid solution held dissolved a large quantity of phosphate of lime and iron, and left on evaporation, silica, probably derived from the crucible. The acid liquid, evaporated to dryness, was again digested in a small quantity of very diluted nitric acid, and filtered. Diluted sulphuric acid gave, with this liquid, a white precipitate, not entirely soluble in potash, because phosphate of lime (derived from the tissues) was precipitated by the alkali from the acid solution. The acid liquid was also precipitated, of a deep greenish-black, by a current of sulphuretted hydrogen gas; and on adding more nitric acid, the sulphuret of iron was removed, and a light-brown precipitate remained, which was sulphuret of lead. A portion of the original liquid was then strongly acidified with nitric acid: sulphuretted hydrogen was passed into it, and a brown precipitate of sulphuret of lead only was now thrown down, the iron being suspended. A portion of the original liquid, nearly neutralized by potash, gave in a few seconds, the brilliant yellow precipitate, in crystalline scales, of iodide of lead. The galvanic test of zinc and platina did not answer, the quantity of lead present being too small. The tests acted clearly and decidedly, leaving no doubt that lead was present in the liver of the female in comparatively large quantity.

This case is interesting in several points of view. The suspicion entertained that the system of this female was impregnated with lead, from the blue line observed in the gums, three days after her admission into the hospital, was fully confirmed by the result of the analysis of the liver. The detection of lead in the tissues also appears to show that this symptom, first pointed out by Dr. Burton, is of great value in diagnosis: for it could not be ascertained with any certainty from this female, or any of those who knew her, that a salt of lead had been taken,—and yet it is impossible to doubt that some salt of lead must have been taken. The other symptoms observed during life, such as vomiting obstinate constipation, and paralysis, also corroborate the view that this was a case of poisoning by lead. The pain in the abdomen, unlike that produced by lead, was, however, increased by pressure. The post-mortem appearances resemble those which have been met with in cases of chronic poisoning by lead. This remark applies more especially to the state of the large intestines, which were found irregularly contracted and distended. There were no signs of irritation or inflammation in any part of the alimentary canal.

Are we to regard this as a case of acute or chronic poisoning by lead? The rapid progress of the case, and the severity of the symptoms after the admission of the female into the hospital, appear to indicate that it was an acute case. The question here arises, however, what is the shortest period, after lead-poison has been taken, within which blueness of the gums may appear? This state of the gums, as it has been already mentioned, was not observed until the third day; and it is unfortunate that there was a very obscure account of the condition of the patient before her admission into the hospital. Blueness of the gums has, I believe been hitherto regarded as a symptom peculiar to the *chronic* form of poisoning; and, so far as I am aware, there are no facts to enable us to state within how short a period in an acute case, this symptom may appear. The absorption of lead, when its salts are taken as poisons, certainly goes on with great rapidity; and the metal is soon found in the organs and secretions; but it may require a much longer time for this effect of the metal to be made apparent by a discolouration of the gums. On the whole, it appears probable, from the severity of the symptoms, that a dose of the poison had been taken only a few days before her admission. The deceased may, however, have taken small doses of sugar of lead some weeks previously, as it was alleged, for the purpose of procuring abortion, under the idea that she was pregnant. To this the chronic gastritis and obstinate constipation might perhaps be referred.

Lead has been of late years so frequently detected in the soft organs and secretions, that the presence of it in the tissues of the body may now be looked for with some certainty, when it cannot be discovered either in the matter vomited or in the contents of the stomach after death. I believe that the liver, from its size and from the large quantity of blood which it contains, is the organ best adapted for analysis. Here, as in the incineration of any of the soft parts of the body, the analyst is liable to be embarrassed by the presence of phosphate of lime and oxide of iron in the ash. The first necessarily renders obscure the solubility in caustic potash of the precipitates of lead, formed on the addition of the tests; and the last gives a colour with sulphuretted hydrogen gas, which, if this test alone were employed might easily lead to a serious error. The sulphurets of lead and iron are, however, very differently affected by nitric acid; and on making the liquid rather strongly acid with this menstruum, we are quite sure that no sulphuret of iron will be formed. In an acid mixture of these two metals, brown sulphuret of lead only is precipitated. This precipitate, however, digested in strong nitric acid, is immediately converted to sulphate and nitrate of lead.

In searching for this metal in the tissues, it is proper to remember that lead may be introduced accidentally into the ash by the crucible, or in other ways which will easily suggest themselves; and as the tests for lead are of exceeding delicacy, it is the more necessary to use extreme caution in the analysis. In the case just related, every precaution was taken to avoid any fallacy; and it will be observed that the three most important tests for lead—sulphuric acid, sulphuretted hydrogen, and iodide of potassium, answered perfectly,—the results of one test thus corroborating those obtained by the use of the others. No traces of metal in a reduced state were procured; but this is not necessary in order to enable a chemist to express a positive opinion of its presence. From the very decided results procured by these tests, lead appears to have been present in the liver in much larger quantity than it is ever found in cases of colica pictonum.

Normal lead.—It has been objected to these processes for the detection of lead, that lead always forms a constituent part of the soft organs in healthy persons. My own experiments agree with those previously made by M. Flandin, in the result that the ash obtained by incinerating the soft parts of the body, does not contain a trace of lead, where care has been taken to exclude the metal during the analysis, and *several tests* have been employed. If sulphuretted hydrogen gas only be used as a test, I can easily imagine that iron would be frequently mistaken for lead.

The quantity of nitric acid and water in which the incinerated ash is digested, should be at a minimum, since the subsequent neutralization of much nitric acid, in a large quantity of water, will materially interfere with the action of the tests. The bulk of the ash may be reduced by finely powdering it, agitating it in a large vessel of water, and pouring off that portion which is suspended. The lead will be found in the heavier particles, which subside instantaneously.

Lead in organic solids.—It is commonly recommended to pour on a portion of the solid a solution of hydrosulphuret of ammonia; and if this be blackened, it is inferred that lead is present. This inference, however, without further experiments, would not be justifiable, since the presence of iron in the solid, might give rise to a similar change. The only certain plan is to burn the organic substance, or to decompose it by heat and to digest the carbonaceous ash in nitric acid slightly diluted. The acid liquid should be filtered and tested by the appropriate tests.

Absorption.—It was formerly a question, whether in cases of poisoning by the acetate of lead, the metal is absorbed and enters into the circulation. It was found by Tiedemann in the blood of poisoned animals, although Flandin failed to detect it in this liquid. Orfila also detected lead in the urine of a female who swallowed an ounce of the acetate (Op. cit. i. 684;) but Dr. Mitscherlich was unable to find any traces of the metal in the blood or urine of animals. The general opinion, however, now is, that the metal is in some form or other absorbed into the system, because after a certain period it is found more or less in all the soft organs, but especially in the liver. During life it appears to be eliminated chiefly by the urine.

GOULARD'S EXTRACT. SUBACETATE OF LEAD.

Goulard's extract is generally seen under the form of a reddish-coloured liquid, as it is often made with common vinegar instead of acetic acid. This substance has caused death in at least four instances,—one in France and three in England. The *symptoms* produced are similar to those described in speaking of the former compound. The subacetate is much more powerful as a poison than the neutral acetate, probably from its containing a larger quantity of the oxide of lead. One fatal case of poisoning by Goulard's extract is marked down in the Coroners' return for 1837-8. In January, 1840, two other cases of poisoning by it occurred in this city in two children, aged respectively four and six years. The quantity taken by the children could not have been very great, but they both died within thirty-six hours. The symptoms were at first violent vomiting and purging;—in one case they resembled those of Asiatic cholera. The bodies were inspected by Dr. Bird, and presented the following *appearances*. The mucous membrane of the stomach was of a grey colour, but otherwise perfectly healthy. The intestines were found much contracted, in one instance more so than in the other. A case is reported by Orfila in which an inspection was made of the body of a man who had been killed by taking a quantity of Goulard's extract. He died within forty-eight hours, and there was well-marked inflammation of the alimentary canal from the œsophagus downwards. The villous coat of the stomach was completely softened, and the effused mucus was found to contain the poison. (Toxicologie, i. 671.) Mr. Marshall mentions a case of recovery where two fluid-ounces of Goulard's extract had been taken by mistake. (On Arsenic, 106.)

Analysis.—The subacetate of lead cannot be so readily procured in a regular crystalline form as the acetate by evaporation. Its solution is strongly alkaline, and it contains a much larger proportion of oxide of lead than the common acetate. The same tests are applicable to it as to the acetate (ante, page 352.) It possesses all the chemical properties of that salt, but differs from it in being copiously precipitated by a solution of gum acacia. The pure solution of subacetate is colourless. That which is commonly sold has a brown colour, owing to its being made with vinegar.

Goulard Water is nothing more than a mixture of one drachm and a half of this solution to a pint of water.

NITRATE OF LEAD.

Dr. Christison found that four hundred grains of this salt killed a dog in sixteen hours. (On Poisons, p. 549.)

Chemical analysis.—The nitrate of lead is commonly seen in solid tetrahedral crystals:—when powdered and heated in a reduction-tube, it gives off nitrous acid vapour, and yellow protoxide of lead is left. It is readily dissolved by water, and the solution is neutral. The nature of its acid is best determined by adding carbonate of potash to the solution,—and filtering:—the filtered liquid should on evaporation yield nitrate of potash. Or a grain of the salt may be dropped into sulphate of narcotine. (See Nitric Acid, ante, p. 189.) All the tests for lead mentioned in speaking of the acetate, apply equally to the nitrate.

CHLORIDE OF LEAD.

Some years since a woman was brought to Guy's Hospital, who had swallowed the chloride of lead: the quantity could not be ascertained, but a portion of the poison was found in the paper, out of which she had taken it. There were no urgent symptoms except vomiting: alkaline sulphates were exhibited, but she suffered so little inconvenience that she left the hospital the same day, and eventually did well.

According to the observations of Mr. Osborne, chloride of lead is likely to exist occasionally in spring water as a poisonous contamination. He states that in the water of some of the wells around Southampton, he found free chlorine. The chloride of lead is soluble in thirty parts of cold water; and when once produced it might give rise to serious symptoms. (Pharm. Times, Sept. 26, 1846, 64.)

Analysis.—This is a white pulverulent salt: when heated in a tube it fuses into a yellowish green mass and remains fixed—it is soluble in hot, but not very soluble in cold water; the solution gives the reactions indicated with the tests for lead: and the chlorine may be discovered by nitrate of silver. This salt is rendered more soluble in water by nitric or muriatic acid;—it is insoluble in alcohol.

Turner's yellow. (**oxychloride of lead.**)—This is commonly seen in the form of a fine yellow-coloured powder; it is sometimes called mineral yellow. Its action on the body is unknown; but it is much used in the arts, and as it might be mistaken for another substance, its chemical properties require a brief description.

Analysis.—It is a very fusible substance, it melts when heated, aud remains fixed; it is partially soluble in caustic alkalies;—when digested in nitricacid, oxide of lead is dissolved, and a milky white chloride of the metal remains.

CARBONATE OF LEAD.

This substance, also known as *White Lead*, *Ceruse*, *Kremser* or *White*, is extensively used in the arts. It is commonly seen in the form of heavy white masses resembling chalk. It is insoluble in water, but still possesses poisonous properties, a decided proof, among numerous other facts, that insolubility does not prevent a substance from exerting a poisonous action on the system. The very small quantity of free acid contained in the gastric secretions, may, it is true, dissolve a portion; but the quantity of carbonate required to neutralize the free acids in the stomach, would probably produce no serious symptoms whatever; since we commonly find it is only in large doses, that this substance acts as an irritant poison.

Symptoms.—A very interesting case of poisoning by the carbonate of lead, was reported in October 1844, to the Westminster Medical Society, by Mr. Snow. A child aged five years ate a portion not so large as a marble, ground up with oil. For three days he merely suffered from pain in the abdomen and costiveness. On the third night, the child became rapidly worse, and there was vomiting. He died ninety hours after taking the poison, having passed some very offensive motions of a greenish-black colour (probably from sulphuret of lead) before he died. The mucous membrane of the stomach was much inflamed, and of a dark-red colour throughout. Poison could not be detected in the contents or tissues of the stomach, or in the matter vomited. It is remarkable that in this case, so small a quantity should have proved fatal without exciting any marked symptoms of irritation in the first instance. There are many fatal cases of poisoning by the carbonate of lead in the human subject, but it has in these instances proved insidiously fatal, by inducing *Colica pictonum*. They are to be regarded as cases of chronic poisoning. The following instance of recovery from a large dose of carbonate of lead, is reported by Mr. Cross. A woman, aged thirty-three, took by mistake for a dose of magnesia, from *six to eight drachms* of carbonate of lead. Five hours afterwards, she was seen by her medical attendant; she was in a cold perspiration, breathing heavily, constantly vomiting, her pulse hard, small, and quick. There was great anxiety of countenance, with dryness of the throat;—a sense of heat in the stomach, with very painful colics. Castor-oil and sulphate of magnesia, with diluted sulphuric acid, were given to her;—the last at frequent intervals. The extensor muscles became paralysed, and the flexors rigidly contracted,—the colics were so excruciating, that the patient generally fainted after each paroxysm. The evacuations from the bowels were of a very dark colour, probably from the action of sulphuretted hydrogen, contained in the intestines, upon the lead. The symptoms abated, but the next day there were nausea and faintness with griping pains. In four days she was convalescent. A somewhat similar case is reported in Casper's Wochenschrift for 1844, in which a man, æt. 20, swallowed by mistake for chalk, between five and six drachms of carbonate of lead. In a few hours it produced all the symptoms of irritant poisoning, thirst, burning pain and incessant vomiting; yet although he was not seen for twenty-four hours after taking the poison, he perfectly recovered in the course of a short time under very simple treatment. His recovery was probably due to the greater part of the carbonate having been ejected by the early vomiting. (Ann. d'Hyg. 1845, ii. 226.) These cases show that the carbonate of lead, although poisonous, is not very energetic. Its action as a poison is probably not greater than that of the acetate; and, so far as observations on the human subject extend, it is less active than the sub-acetate.

A case is related in the Annales d'Hygiène, (April, 1844,) which shows that serious accidents may sometimes happen from the shot used in cleaning bottles being left, and afterwards becoming chemically acted on by the wine or liquid introduced. The practice of thus cleaning bottles is very common in England and also in France; some of the pellets often become fixed in the narrow part of the base of the bottle, and thus escape notice. A person, after having swallowed a few glasses of liquor, suffered from the most violent colicky pains, and all the symptoms of irritant poisoning. Dr. Hanle, who was immediately called, having observed that the liquor remaining in the bottle was very turbid, poured it off for analysis, when he found firmly wedged in at the bottom of the bottle, ten leaden pellets, which had become so completely transformed to carbonate of lead, that there was only a small nucleus of the metal left. So long as the liquor was clear, no accident had arisen from its use; but the symptoms of poisoning appeared immediately when the turbid portion at the bottom of the bottle, containing the salt of lead either suspended or dissolved, was swallowed. (See p. 113, ante.) It is singular that the lead should have been found in this case in the state of insoluble carbonate; for, in general, the vegetable acids contained in wine (if we except the tartaric) form soluble salts of the metal. With acescent wines, such as those made in this country, which owe their acidity chiefly to citric acid, accidents of this kind are very liable to occur; but with good Spanish wines, they are not so common. (See page 367, post.) The acidity here is chiefly due to tartaric acid; and it is only slowly that tartrate of lead is formed, even when the quantity of shot left in the bottle is large. It is very unusual to meet with a case in which this substance is employed with the intention to murder: a woman was, however, lately tried in France for mixing carbonate of lead in flour which was made into bread. Several persons partook of it, and all suffered more or less, the symptoms being general uneasiness, colicky pains, and vomiting, with obstinate constipation. The witness attributed the symptoms to carbonate of lead, but stated that it was not a very active poison, as he had known an instance in which a man had taken five drachms and recovered. Upon this evidence the woman was acquitted. (Journal de Chimie Médicale, 1845, 532.) The quantity of carbonate in this instance, formed about two per cent. of the weight of the flour.

TREATMENT.—It is obvious that the alkaline sulphates could not here be employed as antidotes, since it requires long digestion at a high temperature, for these salts to re-act on the carbonate of lead; and even then the decomposition is only partial. I would suggest, in a case of this kind, the expediency of administering an alkaline sulphate mixed with vinegar or some weak vegetable acid, such as lemon-juice. Emetics and the stomach-pump should also be employed.

COLICA PICTONUM.—PAINTER'S COLIC may be regarded as a *chronic* form of poisoning by carbonate of lead, indicated by violent pain in the bowels, constipation, and paralysis. The carbonate finds its way into the system, among white-lead manufacturers, either through the skin or through the lungs, or both together;—it becomes diffused in a fine powder through the atmosphere, and thus enters into the lungs. It has been remarked in France, that in manufactories, where the powder was ground dry, not only have the labourers suffered, but also horses, dogs, and even rats, have died from its effects. Since the practice has arisen of grinding the carbonate in water, cases of colica pictonum have not been so numerous. They are still, however, frequent among painters, the manufacturers of some kinds of glazed cards, the bleachers of Brussels lace, and among those engaged in the glazing of pottery, where oxide of lead is employed in the glaze.

It is to be regretted that a substance so pernicious to health as the carbonate of lead, should be so extensively employed in the arts and manufactures. Much is said concerning the preventable deaths from bad drainage and defective ventilation, but the yearly deaths from unnecessary poisonings are wholly disregarded. Under a proper system of medical police, precautions would be taken to prevent the wide-spreading of a secret source of disease and death. M. Chevallier has recently called the attention of medical men to this subject. Preparations of lead are used in cases where their presence would hardly be suspected. It appears that in the manufacture of Brussels lace, if it were washed before it became an article of commerce, its value would be seriously reduced. It is therefore whitened, or, as it is called, bleached (!) by sprinkling it with finely powdered carbonate of lead, and placing it between folds of paper. In this state it is well beaten, in order to incorporate the powder with the fibre. The workmen who perform this operation are thus constantly breathing an atmosphere of this poisonous salt, and they suffer from dryness of the fauces, colic, and all the other symptoms of chronic poisoning by lead. (Ann. d'Hyg., 1847, i. 111.) It becomes a question whether those females who wear this lace in close contact with the skin, may not suffer from symptoms of lead-poisoning. Actors who employ carbonate of lead as a cosmetic, to give paleness to the countenance, are known to be liable to attacks of lead-colic. The makers of glazed cards, in which white lead is largely employed, also suffer from this disease. I have lately ascertained that the paper on which many of the finest French engravings are printed, is saturated on its upper surface with carbonate of lead: softness is thus given to the impression, and the defects of a worn copperplate are thereby concealed: but, on the other side, there is the risk of chronic poisoning to which the workmen are exposed, and the certainty that the prints will become tarnished and destroyed by the slow action of sulphur in the atmosphere. From a paper read at the Institute by M. Chevreul, it would appear that the size or composition, used in the manufacture of woollen fabrics, is strongly impregnated with lead, either in the state of oxide or salt. The wearing of these poisoned articles of dress in contact with the naked skin must be productive of injury. So easily is the system affected, that colic and paralysis have been known to arise from an individual working or sleeping in a recently painted room. (Lancet, Oct. 26, 1844.) In a case lately reported by Dr. Chowne, a man who slept in a newly-painted room for a few nights was attacked with paralysis. (Med. Gaz., xxxix. 255.) I have myself suffered from severe colic by respiring the vapour of fresh paint. It is not improbable that, in these cases, the carbonate of lead is carried off in vapour, in combination with that of the essential oil of turpentine.

SYMPTOMS.—The diagnostic symptoms of chronic poisoning by lead are well marked. There is first pain, with a sense of sinking commonly in or about the region of the umbilicus. Next to pain there is obstinate constipation, retraction of the abdominal parietes, loss of appetite, thirst, fœtid odour of the breath, and general emaciation. The skin acquires a yellowish or earthy colour, and the patient experiences a saccharine, styptic, or astringent taste in the mouth. A symptom of a peculiar nature has been pointed out by Dr. Burton (Med. Gaz. xxv. 687,) namely, a *blueness* of the edges of the *gums*, where these join the bodies of the teeth: the teeth are of a brownish colour. Dr. Chowne states that from inquiry and observation, he is satisfied that the presence or absence of this blue line is not connected with the administration or non-administration of lead. (Lancet, Oct. 26, 1844.) It has, however, been so frequently observed, that most pathologists now regard it as a well-marked pathognomonic symptom. A similar blue mark around the edges of the gums has been noticed in other cases of poisoning—as by mercurial preparations (ante, p. 311;) and it is possible that in an advanced stage of chronic poisoning by lead it may be absent, (see a case by Mr. Fletcher, Med. Times, Feb. 14, 1846, p. 395):—as where, for example, the individual has ceased to expose himself to emanations of lead. Many facts tend to show that it is an early symptom. The disease often kills the patient; and after death the large and small intestines are found contracted,—especially the colon. The persons most subject to this form of chronic poisoning, are workers in white lead and painters. Out of 1330 cases received during five years (1838-42) into the Parisian hospitals, 655 were among these classes of workmen. Of 341 who were workers in white lead, 55 died. The workers in metals,—plumbers who handle metallic lead, are but little subject to this disease. Only 22 cases of this kind occurred in the five years. (Gaz. Méd., Janvier 17, 1847.)

Absorption.—M. Grisollé made numerous analyses of the fluids and viscera in fatal cases of painter's colic, without detecting any traces of the metal. The urine of workmen, employed in white-lead manufactories, has been repeatedly examined, but no lead has been discovered in it. This was owing probably to some defect in the analysis, or to the very small quantity absorbed; because the tests for lead are remarkably delicate, and would detect it were it in no larger proportion than the 158,000th part of the liquid examined. Recent researches have shown that the metal is absorbed in some form, probably in all cases of lead-poisoning; and as a singular proof of the absorption, and diffusion of the metallic salt in cases of lead-colic, it may be mentioned that sulphuret of lead is formed in these subjects on the surface of the skin, during the employment of sulphureous baths. This appears to show that the poison is eliminated through the skin. Lead has been discovered by Professor Cozzi in the blood of persons labouring under this disease, and in one case Dr. Inman discovered it in the cerebellum. (Med. Gaz. xxxviii. 389.)

In the following instance of poisoning by paint, I have reason to believe that it existed in the milk. A few years since a cow drank up a large quantity of paint, of which carbonate of lead was the chief ingredient. The animal suffered severely;—sulphate of soda was largely exhibited, and the cow eventually recovered. While the animal was labouring under the effects of the poison, a quart of milk was drawn from it, put into a glass bottle, and sent to me for examination. I found that sulphuretted hydrogen gas gave with it, a minute black flaky precipitate, which appeared to be sulphuret of lead. I tried several other specimens of milk, obtained in the usual way from London dealers, without finding that they were in the least affected by a current of sulphuretted hydrogen gas. (See Guy's Hos. Rep. No. xii. O. S.) This result seems to show that in poisoning by this salt of lead the metal is taken up in some form and excreted.

In the TREATMENT of these cases of chronic poisoning it has been proposed to use sulphuric acid and sulphuretted waters. Experience has however, shown that they are quite inefficacious. (Orfila, i. 686. Galtier, i. 676.)

WATER POISONED BY LEAD.—One other form of poisoning by carbonate of lead is of some interest to the medical jurist,—I allude to the impregnation of *water* with this substance by contact with metallic lead. This metal, it is well known, is largely used for water-cisterns and pipes; and, under certain circumstances, hydrated oxide and carbonate of lead are apt to be formed in large quantity, and to be diffused in the water. It has been supposed that carbonic acid contained in water would partially dissolve, and suspend the carbonate in it; but in saturating water with carbonic acid, over finely divided carbonate of lead, it was not found on filtration that any perceptible portion had been dissolved. This conversion to carbonate is chiefly observed to take place with new lead, or the metal which has a bright and polished surface; but it is remarkable that when lead is alloyed with 1-200th part of arsenic it remains unaffected by water. Old lead does not easily produce the oxide or the carbonate, the metal being protected by the crust which is already formed on it and firmly adheres to it. It is important for the medical jurist to bear in mind that the purer the water or the less saline matter it contains, the more it is liable to acquire this poisonous impregnation; but pure water has no effect whatever unless there be free access of air. When water thus contaminated with carbonate of lead is used, symptoms of poisoning may suddenly affect a whole family, without the cause being at first apparent; or one or more cases of chronic poisoning by lead, may unexpectedly show themselves among the members of a household where such water is drunk. This subject has been most fully examined by Dr. Christison. I have repeated his experiments, and made some others, the details of which will be found elsewhere. (Guy's Hosp. Rep. No. vi. O. S.)

There are two kinds of water liable to become poisoned, when kept in contact with lead under a free access of air. 1. That which abounds in carbonic acid; 2, that which contains little or no saline matter. Of this last we have instances in distilled water, pure rain water, or remarkably soft water. With respect to highly carbonated water, this may be known by boiling a portion in a flask or retort, and conducting the gaseous products into a solution of subacetate of lead. Lime-water will also serve as a test for gaseous carbonic acid, although it is not so delicate. The second kind of water is known by evaporating at least a gallon to dryness, and noting the weight of saline matter obtained. If this form less than the 15,000th part of the weight of the water, it is liable to acquire an impregnation from lead. The Thames water contains about the 7000th of its weight of saline matter; and I have kept in this water for nine years a mass of lead, exposing fifty-eight square inches of surface, without any carbonate being produced. As the water was slowly lost by evaporation, the quantity was made up. Distilled water treated in like manner during the same period, has produced a very considerable quantity of hydrated oxide and carbonate of lead. A mixture of equal parts of river and distilled water had no action on lead; consequently a preservative effect existed where the proportion of saline matter could not have been greater than the 14,000th part. The Edinburgh water, according to Dr. Christison, contains about the 12,000th of its weight of saline matter; and it has but a feeble action on lead. That used at Tunbridge some years since, contained only the 28,000th part, a quantity so small, that with a knowledge of these facts, it is not surprising the water should have become contaminated with lead, and have given rise to lead-colic among the inhabitants. The same fact has been more recently observed in the water of Southampton.

This chemical production of hydrated oxide and carbonate of lead, takes place with extreme rapidity. On putting a fresh-scraped piece of the metal into about three ounces of distilled water, I have observed a milky film to be formed around it, in the clear sunshine of summer, in the course of a few minutes; and in twenty-four hours a thick crystalline sediment of carbonate of lead was deposited. Rain-water collected directly from the atmosphere, had the same property: but that which had passed over roofs of slate or tile, was more slowly, and in some instances not at all affected. This production of hydrated oxide and carbonate, by contact with bright lead, is a very good test of the absolute purity of distilled water; and conversely this experiment is the best that can be suggested, for determining whether any particular kind of water is liable to acquire a poisonous impregnation from that source. If the fresh metallic surface remain bright after some days, or only acquire a faint incrustation of sulphate;—and hydrosulphuret of ammonia does not give a brown tint to the water, there is but little danger of its becoming poisoned with lead. Water which does not acquire a poisonous impregnation by contact with lead, may, however, become poisoned if the vapour which slowly rises from it, comes in contact with the metal. Thus leaden covers to cisterns, or partially filled cisterns, are generally corroded by the pure water which comes in contact with the lead as a result of evaporation.

My experiments have led me to the conclusion, that *Sulphate of lime* is the salt which, by its presence in most kinds of hard water, prevents this action on lead. When this salt forms only the 5,000th part of the weight of water, no carbonate of lead is produced;—and the sulphate of lime, dissolved in this or in a larger proportion in distilled water, will confer on it the properties possessed by river-water. Sulphate of lead appears to be slowly formed; this closely invests the metal, and prevents the production of any oxide or loose crystalline carbonate. Thus then a water, which is abundantly precipitated by a salt of barytes, and by oxalate of ammonia, is, cæteris paribus, not very likely to give rise to lead-colic by passing through leaden pipes, or by being preserved in leaden cisterns. The facts connected with the contamination of water by lead, are of great interest. In a trial which took place at the Surrey Lent Assizes, 1845, (*Solomon* v. *Lawson*, see ante, p. 52, also Law Times, May 16, 1846, 135,) these facts were of some importance, and the effects of water so poisoned, on the system, were closely investigated.

Dr. Christison has observed, that water may contain lead, although perfectly transparent when drawn. The lead may be dissolved in the state of hydrated oxide; this will become converted to carbonate on exposure to air. According to this experimentalist, it is not carbonate of lead which is produced by the continued action of pure water on this metal, but a permanent compound, —formed of two equivalents of carbonate of lead and one of hydrated oxide. Water thus impregnated with carbonate and oxide of lead, may be partially deprived of lead by filtration through animal charcoal. It need hardly be observed, that much saline matter may be present in water, and yet the lead may become rapidly affected; for the salts may not be of a kind to have any preservative property. The deposit which takes place on leaden cisterns is often ignorantly regarded as the result of the hardness of water; it is scraped off, and a fresh surface of metal is exposed; so that in a case of this kind, water from a particular cistern may have been for a long time used with impunity, and yet suddenly give rise to symptoms of lead-poisoning, probably to the surprise of the medical attendant and the parties affected.

ANALYSIS.—Carbonate of lead is a solid white powder, insoluble in water, and immediately blackened by sulphuretted hydrogen or hydrosulphuret of ammonia. 1. When heated on platina, it leaves a residue of yellow or orange-coloured oxide of lead, soluble in nitric acid. 2. The carbonate is easily dissolved by diluted nitric acid with effervescence, which shows that it contains carbonic acid. The oxide of lead, combined with nitric acid, may be readily detected by the tests already mentioned. If the carbonate be mixed with sulphate of lead or sulphate of barytes, these bodies will remain undissolved by nitric acid. The salt may be easily reduced on charcoal by the blow-pipe, and metallic lead procured. In *chronic* poisoning by carbonate of lead, the poison is not likely to be found in the stomach: it exists only in the tissues of the body, and from these it may be separated by the usual process of incineration. (See ante, p. 352.) The presence of hydrated oxide or carbonate of lead in *water*, is immediately indicated by its acquiring a brown colour, on adding to it a solution of hydrosulphuret of ammonia. When the carbonate of lead is suspected to exist in organic *solids*, its presence may be known, not merely by the substance acquiring a dark colour on treating it with the hydrosulphuret of ammonia, but by its becoming of a bright yellow on adding to it a few drops of acetic acid and a solution of iodide of potassium.

SULPHATE OF LEAD.

This salt, owing to its great insolubility, is not commonly considered to be poisonous. It is, perhaps, not more insoluble than carbonate of lead, calomel, or Scheele's green and yet these substances are known to act on the body. There is, however, good reason to believe that its action, if any, is considerably less than that of the other salts of lead: and on this fact, the antidotal treatment of lead-poisoning by alkaline sulphates, is based. M. Dupasquier ascertained that *seventy-seven* grains of the sulphate might be given to a dog kept fasting for twenty-four hours, without exciting vomiting or any other unpleasant symptoms. The dog was kept four days, and the dose produced no effect. On killing the animal, and inspecting the body, there were no abnormal appearances. Doses of 150 and 300 grains were given to other dogs, without producing symptoms of poisoning (Consult Méd. Lég. 1843, p. 15.) Orfila states that he gave to a dog 554 grains in a finely pulverised state, without any injurious effects resulting. The dog ate his food as usual the following day (Op. cit. i. 690.) These, therefore, are strong facts in favour of the sulphate being inert. In a case of acute poisoning, owing to the administration of antidotes, this salt of lead may be found in the stomach scattered in white masses over the mucous membrane. Being very heavy, it will not be difficult to separate it by decantation; and as it is quite insoluble in water and most acids, it will be proper to describe the method of determining its nature.

Analysis.—Sulphate of lead is a white solid, resembling the carbonate by its insolubility in water, but differing from it in not being soluble in acids, except in strong muriatic acid (on boiling) without effervescence. 1. When dropped into the hydrosulphuret of ammonia, it is blackened; and thus it is known from the sulphate of barytes, which it otherwise resembles. 2. When heated on platina-foil, it remains unchanged, or becomes only slightly darkened, if any organic matter be mixed with it, or the flame of the lamp come in contact with it. 3. When suspended in water, and a current of sulphuretted hydrogen is passed through it,—black sulphuret of lead is precipitated (for the analysis of which see ante, p. 353,) and sulphuric acid is dissolved by the water, in which, after evaporation to get rid of the sulphuretted hydrogen gas, it may be detected by the appropriate test, namely, a salt of barytes. Or 4th, Heat the sulphate over a spirit-lamp in a small glass tube with its bulk of cyanide of potassium. It is thus converted to sulphuret. A portion of this may be digested in water and nitric acid, and a solution is obtained in which oxide of lead is indicated by all the tests. Another portion, placed on a glazed card wetted (see p. 174, ante,) indicates that the salt was a *sulphate*, by the production of a *sulphuret*, and the formation of a brown stain of sulphuret of lead on the card.

CHROMATE OF LEAD.

This is a poisonous salt of lead, well known by its brilliant yellow colour. It is to be procured at all colour-shops, and may therefore easily give rise to accidents. I have not met with any instance of poisoning by it in the human subject, but it would doubtless act like the other salts, *i. e.* by producing paralysis or painter's colic when frequently exhibited in small doses, and symptoms of irritation when taken in large quantities. The chromic acid would also operate as an irritant. It is sometimes used for giving a yellow colour to confectionary, and when mixed with indigo, a green. In this state it has produced colic and other alarming symptoms. (See Ann. d'Hyg., 1829, 421.)

Analysis.—The intense yellow colour of this compound distinguishes it from all other substances, except orpiment and sulphuret of cadmium. From these it is well known, by being immediately turned of a greenish black colour by sulphuretted hydrogen or the hydrosulphuret of ammonia. It is quite insoluble in water, but is soluble in nitric acid, in potash (in which respect it resembles orpiment,) and in muriatic acid, which forms with it chloride of lead, and a mixture of muriatic and chromic acids. On boiling this mixture, chlorine is evolved, and green oxide of chrome is set free. When long boiled with carbonate of potash, it forms insoluble carbonate of lead, and soluble chromate of potash.

IODIDE OF LEAD.

This is a crystalline compound of a rich golden yellow colour. But little is known concerning its poisoning properties. In one experiment M. Paton gave nine grains and a quarter to a cat: after four hours no particular effects were manifested. The dose was then repeated. There was no vomiting, but in twelve hours the animal was uneasy, refused all kinds of nourishment,—lost power in its hinder legs,—appeared to suffer from violent colicky pains, and died in three days. There were no marks of inflammation in the alimentary canal, but there was a yellow patch near the pylorus. (Orfila, i. 702.) So far as this experiment will enable us to judge, the iodide of lead is a very active poison.

CHEMICAL ANALYSIS.—This compound is of a brilliant yellow colour. It is soluble in about twelve hundred parts of cold, and two hundred of boiling water. It is also soluble in caustic potash, and in strong muriatic acid. It is decomposed by nitric acid, iodine is set free, and, on evaporating the liquid to dryness, nitrate of lead is obtained as a residue. This may be dissolved in water, and tested. The iodide of lead is used in the form of an ointment. It constitutes one-ninth by weight of this preparation. (UNG. PLUMB. IOD. P. L.)

SULPHURET OF LEAD.

This compound, which is black, is not possessed of any poisonous properties. It may be analysed by a process already described. (See ante, p. 358.)

OXIDES OF LEAD.

The yellow oxide (massicot,) and the brown oxide (peroxide,) are but little known except to chemists. *Litharge* and minium or *Red lead* are, however, much employed in the arts, and have sometimes given rise to accidental poisoning. Liquids used for culinary or dietetic purposes, especially if they contain a free *acid*, are liable to become impregnated with oxide of lead, derived from the glaze of the vessel in which they are kept, and to form poisonous salts. If vinegar be used, acetate of lead may result. Litharge-glaze is also easily dissolved by alkaline or *fatty* substances. The eating of dripping, or the fat of meat, baked in a newly glazed vessel, has thus been known to give rise to slight attacks of colic; while the symptoms were referred by the party to some substances mixed with the food. A case in which the whole of the members of a family were thus poisoned, will be found in the Lancet, (July 4, 1846, p. 27.) When articles of this kind are impregnated with oxide of lead, the fact is immediately known by their being turned more or less of a brown colour by hydrosulphuret of ammonia. All newly glazed vessels yield more or less traces of lead, on boiling in them acetic acid or caustic potash. In this way, the poisonous nature of the glaze may be tested:—the oxide of lead being dissolved by the acid or the alkali. Litharge was formerly much used to remove the acidity of sour wine, and convey a sweet taste. Acetate of lead, or some other vegetable salt of the metal, is in these cases formed; and the use of such wine may be productive of alarming symptoms. Many years since a fatal epidemic colic prevailed in Paris owing to this cause:—the adulteration was discovered by Fourcroy, and it was immediately suppressed. Such wine is known by its being blackened by hydrosulphuret of ammonia.

Snuff has been known to be adulterated with red lead: in one instance this mixture is supposed to have caused death, and in another, it gave rise to alarming symptoms, (Med. Gaz. xxxii. 138; also Ann. d'Hyg. 1831, ii. 197.)

Mr. Scanlan has called attention to the fact, that oxide of lead is sometimes present in distilled water, when leaden pipes have been used for the purpose of condensing the vapour. It appears, however, to be rapidly converted to carbonate, and thus rendered insoluble. (Pharm. Journ., Aug. 1844.) According to Colonel York, hydrated oxide of lead is always formed and dissolved when a bright surface of lead is exposed to air in contact with pure water. This gentleman thinks that it is permanently held in solution; but unless the absorption of carbonic acid from the air be in some way prevented, it is not easy to see why it should not become speedily converted to carbonate, and rendered insoluble. (Pharm. Journ., Dec. 1845, 279.) I have not found any trace of lead in water after filtration, where the water had been for a short time exposed to air. Mr. Phillips has arrived at the same result. (Pharm. Journ., Jan. 7, 1845.) This negative effect has been attributed to the absorbing action of the filter; but this explanation does not appear to me to be satisfactory.

Poisoned wine.—Lead-shot are much employed for the purpose of cleaning wine-bottles, and pellets are frequently left in the bottles. A question has arisen, whether *wine* introduced into them, is liable to acquire a poisonous impregnation from lead. I have found, when the shot are in much larger proportion than could ever be left by accident in a wine-bottle,—that good wine, whether port or sherry, becomes only very slowly impregnated with lead. After two or three months a white sediment had formed, but no lead was dissolved; after thirteen months, the port wine retained its colour, and scarcely any portion of lead was dissolved by it; the sherry had become darker in colour, and the presence of lead was very evident in it. After the lapse of six years, the port wine still had a dull red colour, and gave only faint traces of lead with hydrosulphuret of ammonia and sulphuretted hydrogen gas:—the sherry had acquired a very pale straw colour, and was pretty strongly impregnated with lead. Thus, then, even under the most favourable circumstances, good wine is but slowly contaminated by contact with lead-shot, and white wine more than red. Very acid wines, such as those made from the currant or gooseberry, may, however, become much more rapidly impregnated with the metal, and in a quantity sufficient to produce colic or other serious symptoms.

Cider is apt to become poisoned with the salts of lead when it comes in contact with this metal. It has been generally supposed that the only poisonous compound produced in this case is the insoluble malate; and it appears from an accident which occurred lately in France, where six persons were seized with symptoms of lead-poisoning from drinking cider, that Chevallier and Ollivier discovered that the salt which caused the symptoms was the malate of lead. A large quantity of acid may probably suspend this and other vegetable salts which are reputed to be insoluble; or it may happen that, like the carbonate of lead in water, the insoluble salts may be diffused through the liquid, and suspended in an extreme state of division. In some instances, the carbonate of lead itself may be formed and act as the poison. A case of this kind has been already given. (See ante, p. 359.)

New rum, as it is made in the West Indies, often contains lead derived from the worm of the still, and lead-colic frequently attacks those who drink it. *Old rum*, on the other hand, is by no means unwholesome, and is therefore in great demand. Dr. Traill gives the following explanation of this difference in properties. He found that the rum which was received in glass-bottles from the still, was always impregnated with lead; but when kept in oak-casks, the tannin of the oak is slowly dissolved by the spirit, and precipitates the lead in an insoluble form, the spirit thereby becoming perfectly wholesome. He has suggested that a little decoction of oak-bark, added to the new rum, would render it equally innoxious. (Outlines, 112.)

Sugar.—It has been found that sugar is sometimes the medium of conveying lead-poison into the system, and giving rise to attacks of colic in those who partake of it. Dr. Jackson has reported an instance of this kind, in which several persons lost their lives, and many others were attacked with paralysis and colic, who had partaken of sugar which had probably been kept in leaden reservoirs. Lead was discovered in the sugar in large quantity. (Med. Gaz. xvii. 1036.)

ANALYSIS.—*Litharge* is commonly seen in reddish or yellow-coloured scaly crystals, insoluble in water, but soluble in great part, or if pure, entirely in diluted nitric acid. The solution possesses all the characters of nitrate of lead. Minium or *Red lead* is commonly seen as a rich orange-red powder;—it is partially dissolved by acids,—a portion of brown peroxide being left. The solution gives the usual reactions with the tests for lead. Both of these oxides are easily reduced on charcoal by the aid of a blow-pipe; or by mixing them with paste,—painting with this mixture a piece of card, drying it and burning it, when metallic lead is immediately produced. Minium is known from Vermilion among other properties by its being blackened by hydrosulphuret of ammonia; from Red oxide of mercury, by the action of nitric acid, as well as by the effect of heat. Red oxide of mercury when heated is entirely dissipated into oxygen and mercury,—minium gives off oxygen, but remains fixed as an orange-yellow oxide of lead. Red lead is a common colouring matter in red wafers, or wafer-cake, used for destroying vermin. The brown or peroxide of lead does not often require to be examined. It is converted to yellow protoxide soluble in nitric acid, by boiling it with a few grains of gallic acid.

EFFECTS OF EXTERNAL APPLICATION.—Oxide of lead and the salts of this metal, have been known to affect the system even when applied to the skin. Most *hair-dyes* are composed of a mixture of lime and oxide or a subsalt of lead. (See Ann. d'Hyg. 1832, ii. 324.) The long-continued use of these preparations may give rise to symptoms, for the origin of which a practitioner might not be able to account. Dr. Brück of Hanover observed that a violent ophthalmia was induced in a lady who had used for dying her hair, a substance called *Poudre d'Italie*, which on chemical analysis was found to consist of lead and lime. (Med. Gaz. Nov. 1842.) The facts connected with poisoning by lead or its preparations, applied *externally*, are of some interest. They commonly assume the form of chronic poisoning. Even the pure metal frequently handled may thus find its way into the system, unless strict cleanliness be observed. Among the cases mentioned by Orfila is one of a female, who was in the habit of applying for a long period to her face and neck, a cosmetic containing a preparation of lead. After six months there were the usual symptoms of chronic poisoning (ante, p. 361.) This female ultimately became blind and paralytic, and soon afterwards died. In another instance the symptoms had become so firmly established before the cause was suspected, that no treatment sufficed to relieve them. (i. 680.) Notwithstanding these facts, M. Tanquerel does not consider that serious symptoms can be produced by preparations of lead coming in contact with the unbroken skin. If the skin be abraded, then absorption may take place rapidly. A case is reported by Tauffieb, in which the frequent application of lead-plaster to an ulcer of the leg, was followed in less than three months by all the symptoms of chronic poisoning. (Galtier, i. 698.) The use of simple lead-wash in cutaneous disorders has not been attended with any injurious effects.

Among the pharmaceutical preparations for external use, into the composition of which lead enters, is the Compound ointment of lead (Ung. Plumbi Comp.,) the basis of which is lead-plaster, the other ingredients being chalk, vinegar, and olive oil; the Ointment of iodide of lead (Ung. Plumbi Iodidi,) see ante, p. 366; Cerate of acetate of lead (Ceratum Plumbi Acetatis,) a mixture of acetate of lead, white wax, and olive oil, the salt of lead forming but a very small proportion; Compound lead-cerate (Ceratum Plumbi Comp.,) consisting of a solution of subacetate of lead, wax, olive oil, and camphor,—this is commonly known as *Goulard's cerate;* Lead-plaster (Emplastrum Plumbi,) prepared with oxide of lead, olive oil, and water. Lastly, the Liquor Plumbi Diacetatis, which has been already described under the name of Goulard's extract of lead. (See ante, p. 357.) Any of these preparations may be brought for analysis as poisons; they are regarded as such by a low class of criminals, and may be administered in an attempt to destroy life.

CHAPTER XXVIII.

COPPER—EFFECTS PRODUCED BY THE METAL AND ITS ALLOYS. BLUE VITRIOL. SYMPTOMS. CHRONIC POISONING. POST-MORTEM APPEARANCES—TREATMENT. POISONING BY VERDIGRIS—SUBCHLORIDE OF COPPER—CARBONATE—SCHEELE'S GREEN—CHEMICAL ANALYSIS—TESTS—SPECIAL CHARACTERS OF THE SALTS. OXIDES. VERDITER. COPPER IN ORGANIC LIQUIDS—IN THE TISSUES—IN THE SOIL OF CEMETERIES—IN ARTICLES OF FOOD—SLOW OR CHRONIC POISONING—ACTION OF WATER ON COPPER.

General Remarks.—Copper itself is said to be destitute of poisonous properties; but it would appear that when alloyed with other metals and reduced to a finely pulverulent state, it may act as a poison. A singular instance of this kind occurred a few years since. The printing in gold, as it is termed, is performed by means of a species of bronze or copper alloy. The letters are printed with a mixture of size and gamboge; and the copper alloy, reduced to such a fine state of division that it floats in the atmosphere in an impalpable dust, is then brushed over the surface. A boy employed in this occupation was, on the third day, seized with vomiting of a green-coloured fluid, heat and constriction of the œsophagus, pain in the stomach, loss of appetite and rest, and a severe itching in all those parts which were covered with hair. These on examination were found to be of a deep green colour. The boy soon recovered. About twelve other persons, employed in the same work, suffered from similar symptoms; but this did not prevent them from continuing the work. The poison in this case probably entered the system through the lungs and skin. This peculiar effect of finely divided copper in giving a green tint to those parts covered with hair, is mentioned by Dr. Falconer in his Essay on the Poison of Copper, (p. 45) published in 1774.

An alloy of this kind, made to resemble gold, is largely used in the ornamenting of gingerbread and confectionary. I am not aware of any accident having occurred from its being thus eaten: but it is a dangerous practice, and in France is especially prohibited under a penalty by police regulations. (Journal de Chimie, Fevrier 1847.) This alloy is easily known from gold by its entire solubility in nitric acid, forming a greenish-coloured solution of nitrate of copper.

Copper coins when swallowed may produce a certain amount of poisonous salt from the action of the alkaline chlorides: but the great danger to be apprehended in these cases is that they are liable to cause death by a mechanical action. (See case, ante, p. 21.)

SULPHATE OF COPPER.

All the salts of copper are poisonous. The two most commonly known are the SULPHATE OF BLUE VITRIOL, and the SUBACETATE OF VERDIGRIS. These substances have been frequently taken and administered in large doses for the purposes of suicide and in attempts at murder. In the latter case the attempts has been immediately discovered, owing to the very strong metallic taste possessed by the salt. This would in general render it impossible that the poison should be taken unknowingly. With the exception of these salts, poisoning by copper is usually the accidental result of the common use of this metal for culinary purposes.

SYMPTOMS.—Sulphate of copper has been frequently given for the purpose of procuring abortion. In doses of half an ounce and upwards it acts as a powerful irritant, and in very young children a much less quantity would probably suffice to kill. The salt speedily induces vomiting of the most violent kind; this sometimes effectually expels the poison from the stomach, and the person recovers. The *vomited* matters are remarkable for being of a *blue* or *green* colour; and broken crystals of blue vitriol have been discovered in them when the poison was taken in a loosely pulverulent state. If the green colour of the vomited liquids be owing to altered bile, it will not acquire a blue tint on adding to a portion of the liquid, a strong solution of ammonia. If a salt of copper be present, this change of colour will serve to indicate the fact. There is headach, pain in the abdomen, with diarrhœa; the pain is of a colicky character; and in aggravated cases there are spasms of the extremities, and convulsions. Dr. Perceval met with a case where the most violent convulsions were produced in a young female by two drachms of the sulphate of copper. Paralysis, insensibility, and even tetanus, have preceded death, when the poison was administered to animals. Among the symptoms casually met with in the human subject, may be mentioned jaundice. This has been observed to attend poisoning by the sulphate, as well as by Scheele's green. The medicinal dose of sulphate of copper as an emetic is from five to fifteen grains, and as a tonic from one to three or four grains.

A woman swallowed rather more than *five drachms* of sulphate of copper. Emetics with albumen were freely given, but with little benefit. After thirty-six hours the pulse was small and the face contracted. There was general uneasiness, with complete suppression of urine. Stimulants were then exhibited in the form of wine and tincture of canella. This treatment appeared to do good, and the woman completely recovered in ten days after she had taken the poison. (Journal de Chimie, 1847, 331.)

There are but few instances in which this poison has proved fatal in the human subject. In 1836, a girl, sixteen months old, put some pieces of *Blue stone* (sulphate of copper) which were given her to play with, into her mouth. In a quarter of an hour, the child vomited a bluish-green coloured matter, with pieces of sulphate of copper in it; the skin was alternately cold and hot, but there was neither diarrhœa nor convulsions. The child died in *four hours*, and was insensible before death. (Med. Gaz. xviii. p. 742.) The coroner and jury did not consider it necessary that an inspection should be made; and yet in the event of murder being committed by the administration of this substance, it would be somewhat unreasonably expected that the medical witness should be fully acquainted with the post-mortem appearances produced by it!

Chronic poisoning by copper.—When the symptoms of acute poisoning have passed away, when the cupreous salt has been taken for a long period in small doses, or the individual has been exposed to emanations from copper-salts, or alloys, other effects are manifested. Sometimes in acute poisoning the patient recovers rapidly, at others he may not be convalescent for a month. The most prominent after-effects are excessive irritability of the alimentary canal, attended with frequent vomiting, colic, diarrhœa, and tenesmus: and there is at the same time great prostration of strength, with emaciation, tremors of the limbs, and occasionally paralysis. These symptoms are witnessed in those who have for some time taken small portions of copper with which their food has become accidentally impregnated.

French pathologists have described a copper-colic to which workers in this metal are liable, owing, as it is supposed, to the inhalation of the fine dust of copper or its oxide. According to Orfila, it is in some respects analogous to lead-colic, but it differs from it in being accompanied by a greater degree of irritation in the alimentary canal. (Toxicologie, i. 912.) ANALYSIS, p. 377.

[For a further account of the symptoms and appearances in slow poisoning by copper, see post, p. 884, COPPER IN ARTICLES OF FOOD.]

POST-MORTEM APPEARANCES.—In poisoning by the salts of copper, the mucous membrane of the stomach and intestines has been found more or less thickened and inflamed in the few fatal cases which have been hitherto examined: the membrane has been found also eroded and softened in poisoning by verdigris. The œsophagus has presented an inflammatory appearance. In a case of poisoning by Verdigris quoted by Orfila, the stomach was found inflamed and thickened, especially towards the pylorus, the orifice of which, from the general thickening, was almost obliterated. The small intestines were throughout inflamed, and perforation had taken place, so that part of the green liquid was effused into the abdomen. The large intestines were distended in some parts and contracted in others, and the rectum was ulcerated on its inner surface. (Toxicologie, i. 623.) The lining membrane of the alimentary canal is often throughout of a deep green colour, owing to the small particles of verdigris adhering to it. It has been said that this is an uncertain character of poisoning by copper; since a morbid state of the bile often gives a similar colour to the mucous membrane of the stomach and duodenum. This objection cannot apply, where the green colour is also found in the œsophagus, and throughout the intestines: and, under any circumstances, the evidence from the presence of a green colour would amount to nothing in the judgment of a prudent witness, unless copper were freely detected in the parts so coloured. It is well to remember, that the green stain, if due to copper, would be turned blue by ammonia. In death from arsenite of copper, the inflammatory appearances would probably be more strongly marked.

VERDIGRIS. SUBACETATE OF COPPER.

This salt produces symptoms somewhat similar to those caused by the sulphate. There is a strong styptic metallic taste, with a sense of constriction in the throat, followed by severe colicky pains,—vomiting of a green-coloured liquid, diarrhœa, and tenesmus. In a case reported by Pyl, a woman who took *two ounces* of verdigris died in three days:—in addition to the symptoms above described, there were convulsions and paralysis before death. Niemann relates that a female, aged twenty-four, swallowed *half an ounce* of verdigris, and died under symptoms of violent gastric irritation in sixty hours. (Taschenbuch, 458.) In consequence of the great uncertainty of its operation, subacetate of copper is not employed internally.

One case of poisoning by this substance is reported in the Edinburgh Medical and Surgical Journal for July, 1844. A woman, aged twenty-eight, swallowed a large dose of verdigris. She was soon afterwards seized with great anxiety, vomiting, acute pains and swelling of the abdomen, sensation of burning heat in the throat, coldness, and severe cramp in the extremities, a labouring pulse, swelling of the face, with the eyes sparkling. An emetic brought away some half-digested food, without any traces of poison. The next morning there was painful deglutition, swelling of the throat, the abdomen tympanitic and painful on the least pressure, the countenance heavy, the face flushed, and the pulse oppressed. About two pounds of a distinctly-greenish fluid, with some blood, were ejected. The symptoms became aggravated; the face and eyelids swollen and red, the eyes prominent, the abdomen flattened, and the rectum so irritable and painful that enemata could not be administered. On the second day there was a tendency to coma, the face was pale, the lips swollen, the gums ulcerated and there was an abundant discharge of viscid saliva, A copious stool was passed—the first since the poison was taken; and acetate of copper was detected in it in pretty large quantity. There were several spasmodic fits. On the third day some viscid glairy matter, of a greenish colour and tinged with blood, was vomited, and the spasms continued. On the fourth day bleeding from the nose with general cramps came on, and the urine and fæces were suppressed. There was coldness of the surface, with convulsions. After the lapse of about a week the patient still had vomitings of greenish glairy matters, with uneasiness in the abdomen: but from this date she gradually recovered. This case is interesting from the course of the symptoms being accurately noted; and it is worthy of remark, that icterus, which some have regarded as a symptom of cupreous poisoning, was at no time present. It is unfortunate that the quantity of verdigris swallowed, was not known. ANALYSIS, p. 378.

SUBCHLORIDE OF COPPER.

This is a rich green compound, known as Oxychloride or BRUNSWICK GREEN, which is formed when common salt has been used in a copper-vessel, and has thus given rise to accidental poisoning. It is also used as a pigment. The following is a case of poisoning by it reported in Henke's Zeitschrift der S. A. i. 188, 1844. A boy between two and three years of age swallowed part of a small cake of green water-colour, such as is sold in the colour-boxes for children. Very soon afterwards he was attacked with vomiting and coldness of the extremities. Notwithstanding the exhibition of an antimonial emetic, the symptoms continued to become aggravated, and the child died. On opening the body, there was nothing to indicate especially the action of an irritant poison, except a slight congestion in the cerebral vessels. The child, it appears, had swallowed about a scruple of the green colour, which, on analysis, was proved to be the common subchloride of copper. It was remarkable that there was not the least sign of irritation or inflammation in the alimentary canal. Death was ascribed to the exhaustion resulting from violent vomiting; and congestion of blood in the brain thereby produced. This case, the details of which are rather imperfectly given, shows that the subchloride of copper is a very active poison, and that it may cause death without leaving any signs of irritation in the alimentary canal. It is to be remembered that it is this compound of copper which is often formed in culinary utensils, and which thereby gives rise to accidents, when any food containing *common* salt has been prepared in the vessel without proper precautions. (See Journal de Pharmacie, Juin 1845, 471.)

Another instance is related by Prof. Barzellotti, in which he himself narrowly escaped partaking of the poisonous food. At a monastery near Sienna, the monks were one day, soon after dinner, seized with violent symptoms of irritant poisoning. They suffered chiefly from severe pain in the abdomen, nausea, difficulty of passing urine, spasms of the muscles, and trembling of the limbs. Those who were affected with vomiting and purging, were speedily relieved; but others, who had no evacuations, suffered from vertigo, cephalalgia, intense thirst, and an unpleasant taste in the mouth. Remedies were applied, and they all eventually recovered. It appeared, on inquiry, that the monks were in the habit of keeping their salt-fish in the copper vessel, in which it was dressed for a second day's meal. This vessel was badly tinned; and when the fish was examined, it was found covered with a green jelly, and the sides of the vessel with which the fish was in contact, had a green colour. The cause of the symptoms was no longer doubtful:—subchloride of copper had been here formed by the action of the salt on the metal. (Quest. di Med. Leg. ii. 185.) Several cases of a similar kind are reported by Orfila, i. 619. ANALYSIS, p. 378.

CARBONATE OF COPPER.

A case of poisoning by this substance has been lately reported by M. Desgranges of Bordeaux. A man died in about six hours, as it was supposed, from the effects of an unknown quantity of this poison which he had taken. When first seen he was comatose; he had sustained some violence from a fall, and there was great coldness of the extremities. There was neither vomiting, purging, nor pain in the abdomen on pressure. On inspection, the œsophagus and stomach were covered with a green-coloured substance. The larger extremity of the stomach was vascular, and the mucous membrane corroded in patches. The mucous membrane of the intestines, as well as the liquid contained in them, was green. Carbonate of copper was found in the stomach, and traces of that metal existed in the urine—none was found in the blood. (Med. Gaz. xxxi. 495.) It is remarkable that in this case there should have been neither vomiting nor diarrhœa. The poison seems to have acted more like a narcotic than an irritant. ANALYSIS, p. 379.

PHOSPHATE OF COPPER.

This is a blue compound occasionally met with in the arts. According to the experiments of M. Leportier, although insoluble in water, it is perfectly soluble in the mucous and acid secretions of the alimentary canal, and may thus exert a poisonous action. Small doses of it, given to dogs, occasioned violent vomiting in about a quarter of an hour. (Ann. d'Hyg., 1840, ii. 110.) ANALYSIS, p. 379.

SULPHURET OF COPPER.

This is a black substance, which appears to possess no poisonous properties unless it has been exposed for a long time to the air. Under these circumstances it becomes partially converted to sulphate, and then acts as a poison. Its analysis will be given hereafter, in the description of the process for separating copper from organic mixtures (p. 380.)

ARSENITE OF COPPER.

This compound, which is known under the names of SCHEELE'S,—EMERALD or MINERAL GREEN, is extensively used as a pigment in the arts; it is also improperly employed to give a green colour to wafers and articles of confectionary. Dr. Geoghegan informed me that an accident occurred in Dublin in 1842, by which fourteen children suffered from symptoms of poisoning in consequence of their having eaten some confectionary ornaments coloured with Scheele's green. In two or three of these cases jaundice followed. This is one of the most active of the cupreous poisons, notwithstanding its perfect insolubility in water; but its effects are rather due to arsenic than copper. The dangerous practice of using this powerful poison to give a colour to confectionary is very prevalent, and accidents often arise from this cause. An instance has been communicated to me, in which three lives were nearly sacrificed at a school near Manchester, owing to the boys having eaten some ornamented confectionary, which owed its green colour to arsenite of copper. They suffered from violent vomiting, severe pains in the stomach and bowels, and spasms in the extremities. Three animals which ate of the vomited matters were attacked by similar symptoms. It is much to be regretted that there is no medical police established by law to restrict the free sale and use of this and other deadly poisons. (See Ann. d'Hyg. 1843, p. 358.) In England poison is allowed to be sold like sugar or starch; and every child is assumed by the law to be capable of protecting himself! If death ensue from such a cause, we find that a coroner's inquisition and a trial for manslaughter take place, to investigate an event which, under simple medical-police regulations, would not have occurred. Even on the continent, accidents from this cause sometimes occur. M. Chevallier relates a singular case in which arsenite of copper was used by a pork-butcher for ornamenting a boar's head supplied at a breakfast given on a festive occasion by an eminent Parisian lawyer! The head was decorated most artistically with masses of fat, which were coloured red and green. One of the guests, well acquainted with chemistry, was struck with the rich green colour of the fat, and reserved a portion for examination. He found the colouring matter to be pure arsenite of copper, forming about two per cent. of the weight of the fat! It appeared on inquiry, that notwithstanding the police-regulations, the butcher's boy had procured the poisonous compound at a neighbouring colour shop. (Journal de Chimie Médicale, Janvier 1847, 16.)

Among the reported cases of poisoning by this substance, are the following. A child, aged three years, swallowed a small capsule of Scheele's green, used by his father as a pigment. In half an hour he complained of violent colic; there was frequent vomiting, with diarrhœa, cold sweats, intense thirst, and retraction of the parietes of the abdomen. The mouth and fauces were stained of a deep green colour. Hydrated sesquioxide of iron was given; in about an hour the vomiting and diarrhœa ceased, and soon afterwards the thirst and pain in the abdomen abated. The next morning the child was well. In another case, a child, a year old, ate several pieces of a cake of arsenite of copper, used for colours. There was immediate vomiting, the liquid containing green-coloured particles of the arsenite, but there were no other urgent symptoms. White of egg with sugared water was given to it. After a short time the child became pale, and complained of pain in the abdomen; the pulse was frequent, the skin cold, and there was great depression. Copious diarrhœa followed, soon after which the child recovered. (Galtier, i. 636.) These two cases of recovery serve to show the fallacy upon which the doctrine of antidotes is founded. In both the recovery must be ascribed to the vomiting; as, if the hydrated sesquioxide of iron be admitted to have exercised an antidotal power in the first case, then white of egg and sugared water acted equally as an antidote in the second case!

The arsenite of copper is much used by painters and paper-stainers, and may under these circumstances give rise to accidents, as the following case will show. A young man, after having been engaged for nine days in printing with this arsenical green, was seized with coryza, swelling of the lips and nostrils, and headach. The next day he experienced severe colic, and great muscular weakness; but these symptoms disappeared in about eight days. It is probable that, in this case, the arsenite of copper had been taken into the body in the state of fine powder.

From a case reported by Dr. Martin, it would appear that this arsenical green may have an injurious effect upon those who inhabit apartments recently painted with this substance. Four pounds of Scheele's green, mixed with milk and lime, had been used in painting the walls of a low damp room. In a few days a putrescent and highly disagreeable odour was perceptible. When the windows were closed, those who remained in the room experienced cephalalgia, pain in the chest, and other disagreeable symptoms. The colour was scraped from the walls, and the room was then inhabited without any of these unpleasant effects being observed. Dr. Martin attributed the effects to the formation of arsenuretted hydrogen, by a reaction of the elements of the milk on the arsenite of copper. The poisonous salt may, however, have been itself carried off in vapour by the volatile oil of turpentine. It is in this way, probably, that the carbonate of lead is volatilized, and affects persons who breathe the atmosphere of a room which has been recently painted. A few square feet of painted canvass in an artist's studio, unless well ventilated, may thus produce serious symptoms.

In a note attached to Dr. Martin's case, it is stated that since the mixed acetate and arsenite have been substituted for carbonate of copper in painting the walls of rooms, many persons who have slept in rooms painted green, have complained in the morning of headach, nausea, dryness of the mouth and cough. The symptoms have gone off during the day. In one instance the foul odour was referred to mice, and the wainscot was about to be removed, when a suspicion arising that it was owing to the green colour used as a pigment, this was removed, and the smell disappeared. (Gaz. Méd., 13 Feb. 1847, 130.) So far as I know, no accidents from this cause have occurred in England. (ANALYSIS, p. 379.)

EFFECTS OF EXTERNAL APPLICATION.—The salts of copper are capable of acting locally, and if applied to a wounded or ulcerated surface, they may become absorbed, and seriously affect the system. Sulphate of copper is occasionally used as an escharotic. The solution of this salt, after frequent contact, hardens the unbroken skin, discolours it, and impairs its sensibility. Orfila found that two drachms of acetate of copper, finely powdered, when introduced beneath the cellular membrane of the neck of a large dog, caused death in five days. In another experiment, the same dose applied to the cellular tissue of the thigh, killed the animal in thirty hours. (i. 618.) Violent phlegmonous inflammation is sometimes occasioned by small quantities of the salts of copper becoming introduced into the system through wounded or abraded surfaces. Mr. Stafford met with a case in which a woman pricked her thumb with a pin. She afterwards scoured out a dirty copper, and her thumb immediately swelled to double its natural size. The whole hand and arm became much swollen and inflamed, and extensive abscesses formed: the patient also suffered from fever, from which she slowly recovered. A second case occurred to the same gentleman, in which severe symptoms followed the puncture produced by corroded copper wire. (Med. Gaz. xxxv. 828.) In these cases the poisonous salt may be the carbonate, subacetate, or subchloride,—most commonly the former. It is probable that the severity of the symptoms may be in some instances ascribable to peculiarity of constitution,—the very small quantity of the salt of copper which can be absorbed, scarcely sufficing to account for them.

TREATMENT.

In general there is violent vomiting,—the salts of copper acting powerfully as emetics. The efforts of the stomach should be promoted by the free exhibition of warm water, milk or any mucilaginous drink, and the use of the stomach-pump. This instrument is of little service, when the poison has been taken, as it generally is, in coarse powder. Various antidotes have been proposed. *Sugar* was formerly strongly recommended, on the principle that it had the property of reducing the salts of copper to the state of insoluble red oxide; but this is only under very peculiar circumstances, not likely to be met with in the stomach. (Annales d'Hyg. 1833, ii. 207.) M. Postel is still inclined to regard it as an antidote, although it seems that animals to which he administered it died; but not *so rapidly* as when the poison was allowed to act by itself! The *protosulphuret of iron* has been lately employed as an antidote, and the following case is quoted as an instance of its successful employment; although the quantity of poison taken was unknown. A man swallowed verdigris in some wine, which on examination was found to be saturated with acetate of copper. In a few minutes he was attacked with vomiting, colic, diarrhœa; his pulse was small; there was severe headach, and cold sweats. Two spoonfuls of protosulphuret of iron were given to him every half hour; with albuminous liquids. The vomiting and other symptoms abated; and in three days the man left the hospital quite recovered. (Galtier, i. 634.) Purified animal *charcoal* has been recommended, owing to the property which it is known to possess of separating some metallic salts from their solutions. My experiments with this substance are decidedly adverse to its alleged antidotal properties. Five grains of sulphate of copper were dissolved in five drachms of water, and the solution was shaken with twenty grains of animal charcoal, and filtered after forty-eight hours. The filtered liquid contained to all appearance as much cupreous salt dissolved as before the experiment. When the proportion of purified charcoal was twelve and even thirty times that of the sulphate, the filtered liquid still gave all the reactions for copper. Vegetable charcoal is still less efficacious. It was found to require sixty parts of vegetable charcoal to remove one part of sulphate of copper. When thirty parts were employed, oxide of copper was still present in solution. *Albumen* is well known to form an insoluble compound with oxide of copper, provided the albumen be in very large excess; for the albuminate of copper is easily dissolved by an excess of the solution of sulphate. How far this would act on the insoluble part of verdigris (Subacetate) it is difficult to say; as also whether the albuminate be not itself a poison; still it may reduce the activity of the soluble salts of copper, and thus it is advisable to administer albumen both of the yolk and white of egg, conjointly with the other means recommended. Dr. Edwards, some years since, recommended the use of *iron filings* for precipitating the copper; but the action in this case is too slow, and is immediately arrested by the iron becoming enveloped by a thin film of copper. If the iron even precipitated all the copper in the metallic state, sulphate of iron would be formed in the stomach, and this is itself an irritant. The hydrated oxide of iron has been used in poisoning with arsenite of copper. In a case just mentioned (p. 374,) as well as in another where a child swallowed a small quantity of green paint containing arsenite of copper, milk was given, and afterwards the hydrated oxide of iron. In five hours the vomiting had abated, and the child recovered. (Med. Gaz. xxxi. 270.) The recovery was most probably due to the vomiting, and not to the effect of the supposed antidote; for if arsenite of iron were really formed by a reaction of the hydrated oxide on the insoluble arsenite of copper, the former has been proved to be just as insoluble and as poisonous as the latter.

CHEMICAL ANALYSIS OF THE SALTS OF COPPER.

The salts of copper are generally known by their colour: whether in the solid state or in solution, they are either blue or green. The salts of one other metal are also of a green colour, namely nickel; but there are striking chemical differences between the salts of this metal and those of copper. There are *three* very *soluble* salts of copper; two of these are blue, the sulphate and nitrate,—and one green, the chloride. The salt should be dissolved in water, diluted, and the following tests may be then applied. The solutions of the cupreous salts generally have an acid reaction. 1. *Solution of ammonia:* this gives, in a solution of copper, a blueish-white precipitate, which is soluble in an excess of the test, forming a deep violet-blue solution. 2. *Ferrocyanide of potassium*, a rich claret-red precipitate;—if the quantity of copper be small, the liquid acquires merely a light red-brown colour; if large, the precipitate is of a gelatinous consistency. The ferrocyanide of potassium will act on the violet-blue solution produced by ammonia, provided it be much diluted, or an acid added (sulphuric) to neutralize the ammonia. One portion of liquid may thus be tried by the two tests. 3. *Sulphuretted hydrogen gas*, or hydrosulphuret of ammonia, gives a deep chocolate-brown precipitate, or if the copper be in small proportion, merely a brown colour, even in an acid solution. 4. A slip of *Polished Iron* (a common needle) suspended by a thread in the liquid, is speedily coated with a layer of copper, even when the salt is in very small proportion. When much diluted, a drop of diluted sulphuric acid may be added. If the needle be left for some days in the liquid, the iron will be slowly removed, and a hollow cylinder of metallic copper will remain. This may be dissolved in diluted nitric acid, and tested with the foregoing tests; or the needle coated with copper, may be immersed in ammonia and exposed to air. The liquid then becomes slowly blue. Half a grain of sulphate of copper dissolved in sixteen ounces of water, may be thus easily detected. It was proposed by Orfila to substitute *Phosphorus* for polished iron. This substance most effectually separates metallic copper from its salts, as we shall see presently in treating of copper in organic liquids.

5. *The Galvanic test.*—If a few drops of the copper-solution be placed on platina foil,—slightly acidulated with a diluted acid, and the platina be then touched through the solution with a thin slip of zinc, metallic copper, of its well-known red colour, is immediately deposited on the platina. When the quantity of copper is small, there is merely a brown stain; but a blue liquid is formed by pouring on it ammonia.

Delicacy of the tests.—Among these tests, the ferrocyanide of potassium and sulphuretted hydrogen gas produce a marked action on a quantity of the cupreous salt, in which polished iron has no apparent effect. I have found that ammonia fails to indicate with any certainty less than the 100th part of a grain of sulphate in one fluid-drachm of water; but the ferrocyanide of potassium and hydrosulphuret of ammonia produce an evident effect on a solution containing only the 250th part of a grain of sulphate in half a drachm of water. The iron-test failed to detect the 150th part of a grain in a fluid-drachm of water. It is, however, sufficiently delicate for most practical purposes. The galvanic test is not so delicate as the iron test.

Objections to the tests.—Ammonia produces in a salt of *Nickel* a colour somewhat similar to that produced in a salt of copper; but ferrocyanide of potassium precipitates a salt of nickel of a pea-green colour,—a reaction very different to that produced on a salt of copper. The persalts of *Uranium* give with the ferrocyanide of potassium a deep red-colour, which in a diluted state, might be mistaken for the effect produced by copper; but ammonia gives a yellow precipitate in a persalt of uranium, and sulphuretted hydrogen and hydrosulphuret of ammonia give a yellow-brown precipitate of persulphuret of uranium. The colour of the copper precipitate approaches a dark-crimson; that of uranium, a blood-red. When ammonia is added to the two, the uranium precipitate is dissolved, and a yellow deposit of oxide of uranium takes place in the liquid. When ammonia is added to the forrocyanide of copper, it is not entirely dissolved, and the liquid acquires a blueish-green colour. In an *organic* liquid containing no copper, I have seen a pink-red colour produced by this salt; but the liquid test here described, should not be applied to solution of the salts of copper in *organic* liquids. To the action of the third test, when taken by itself, there are many objections; but these are entirely removed by the application of the other tests. The action of iron, and of zinc with platina, is peculiar. It is true that there is one other metal of a *red* colour like copper, namely, *Titanium;* but this is not precipitated by iron, or zinc and platina, in its metallic state. We may now briefly advert to the specific characters of the different salts.

SULPHATE OF COPPER. (BLUE VITRIOL. ROMAN VITRIOL. BLUE STONE.)—This salt is met with in rhombic masses, transparent, and of a rich colour. When reduced to powder it is nearly white, but becomes again blue on melting or dissolving it. It is soluble in four parts of cold and two of boiling water; and is easily obtained in well-defined rhombic crystals by evaporating a small quantity of the solution on a slip of glass. The powder undergoes no change on adding sulphuric acid. Nitrate of barytes added to the solution, indicates the presence of sulphuric acid (page 174, ante.)

AMMONIA-SULPHATE.—This forms a rich violet-blue solution, and is known from the sulphate by its alkaline odour and reaction, as well as by producing a green precipitate with a solution of arsenious acid. The sulphate is acid, and is unaffected by a solution of arsenious acid. Ferrocyanide of potassium gives the characteristic red-coloured precipitate when the ammonia is neutralized by sulphuric acid.

NITRATE.—This salt is crystallized in prisms of a deep blue colour; it is very deliquescent,—extremely soluble in water, and the solution is not precipitated, if pure, by nitrate of barytes or nitrate of silver. When the powdered crystals are mixed with tin filings and moistened with water, nitrous acid fumes are evolved. By adding carbonate of potash to the solution and filtering, nitrate of potash is obtained in the filtered liquid, and the acid may be thereby identified.

SUBACETATE. DIACETATE. (ARTIFICIAL VERDIGRIS.)—There are several varieties of this salt, some of which are blue, and others green. Verdegris is partially soluble in water, as a sesquibasic acetate; but if this be acidulated with acetic or muriatic acid, a solution is immediately obtained, to which the tests for copper may be readily applied. If a portion of the powder be heated in a reduction-tube, a film of metallic copper is produced,—and acetic acid vapour escapes. Acetic acid is, however, readily discovered by boiling the powder in diluted sulphuric acid. Sulphate of copper is at the same time produced, which admits of a ready analysis.

CHLORIDE.—This is seen in deliquescent crystals of an emerald green colour. It is very soluble in water, forming a deep green solution, if concentrated; but becoming blue when diluted. This diluted solution has the remarkable property of becoming green when heated to 212°, and again blue on cooling. It yields an abundant white precipitate with nitrate of silver, insoluble in nitric acid, by which it is easily known.

The *insoluble* or partially soluble salts of copper, which may give rise to questions of poisoning, are the subacetate, subchloride, carbonate, phosphate, and arsenite. They possess these common characters,—that 1, when rubbed on a steel spatula with a few drops of diluted sulphuric acid, metallic copper is abundantly precipitated on the *iron*;—and 2, when dropped into a strong solution of *ammonia*, they acquire a rich violet blue colour. The dried arsenite of copper undergoes this change very slowly.

Subchloride. (Oxychloride. Brunswick green.)—This compound is insoluble in water; but it is easily dissolved by nitric or muriatic acid, and the acid solution will give all the reactions for copper. The simplest way of analysing this salt, is to boil it in caustic potash:—when black oxide of copper will be separated. This may be washed, dissolved in an acid and tested, while the chlorine may be detected in the filtered alkaline liquid on acidulating with nitric acid and adding nitrate of silver. This test will also detect the chlorine in the nitric acid solution of the subchloride.

Carbonate. (Natural Verdigris.)—This is a blueish green compound, which is produced in firm crusts, when copper, brass, or bronze is exposed at the same time to the action of water and air. It is often called *natural* verdegris, to distinguish it from the subacetate or *artificial* verdigris. When heated on platina-foil, carbonic acid is evolved, and black oxide of copper is left. It is insoluble in water; but is dissolved by acids with effervescence, a character which distinguishes it from the other insoluble salts. The acid solution gives the usual reactions with the tests for copper.

Phosphate of copper.—This salt, which is of a blue colour, is dissolved by nitric acid without effervescence. The tests for copper may be applied to the solution.

Oxides of copper. (Mineral green.)—There are two oxides, one red and the other black. According to the experiments of M. Leportier, they are both poisonous, although quite insoluble in water. Their poisonous action appears to depend on their ready combination with the mucous and acid secretions of the stomach. When metallic copper is swallowed, colicky pains and other symptoms sometimes follow in consequence of the metal becoming partially oxidized and dissolved. The experimental researches of M. Leportier show that the pure metal is not poisonous (Ann d'Hyg. 1840, ii. 99;) but it may cause death as a mechanical irritant. (See ante, p. 20.) The oxides may be analysed by dissolving them in nitric acid and applying the tests for a solution of copper. Both give a blue solution with ammonia when exposed to the air, the red oxide slowly.

The only form in which oxide of copper is met with in the arts is under the name of Mineral Green and Verditer. Mineral Green is a name sometimes given to Arsenite of Copper. The compound sold to artists under this name, is, however, commonly a mixture of hydrated oxide of copper and lime in the state of carbonate. It is easily analysed by digesting it in nitric or muriatic acid, which dissolves both the oxide of copper and lime; the former is separated from the latter by a current of sulphuretted hydrogen gas.

Verditer is said to be a mixture of carbonate and hydrated oxide of copper;—it is, however, more commonly hydrated oxide mixed with lime, potash, and alumina. It is of a rich blue colour, which it owes to the presence of a small quantity of muriate of ammonia: when long kept it becomes greenish-blue. The oxide of copper may be dissolved out of it by diluted acids. This colour is largely employed in paper-staining; but we do not hear of accidents from its use.

Arsenite of Copper. (Scheele's Green.)—This salt is of a green colour, the depth of which is modified by the quantity of oxide of copper present. It is insoluble in water, but soluble in ammonia and in acids, forming a blue solution. When very gently heated in a reduction-tube, arsenious acid is sublimed in minute octohedral crystals. These may be collected, dissolved in water, and tested in the usual way:—the residuary oxide of copper may then be dissolved in nitric acid and tested. With charcoal powder, the arsenite gives, although with some difficulty, a ring of metallic arsenic: but the arsenical nature of the salt is easily determined by boiling it with diluted muriatic acid and a slip of metallic copper or copper gauze. (See Reinsch's Process, ante, p. 286.) Metallic arsenic is immediately deposited on the copper. When the arsenite of copper is used in confectionary, the substance upon which it is spread is either soluble (sugar or starch) or insoluble (plaster of Paris.) In either case we scrape off the green colour and digest it in a small quantity of water. In the first case the arsenite of copper is deposited, while the sugar or starch is dissolved: in the second, the arsenite of copper is deposited with the sulphate of lime. The former may be separated from the latter by ammonia, and reobtained pure by evaporation. Should the arsenite be mixed up with fat or oil, it will easily subside as a sediment on keeping the substance melted, and the deposit may be freed from any traces of fat by digesting it in ether. There is another kind of green pigment much used, called Schweinfurth green. This is a mixture of arsenite and acetate of copper. The presence of arsenic in this compound is easily detected by muriatic acid and metallic copper. The arsenite of copper has been placed among cupreous poisons, because it so closely resembles them in physical and chemical properties;—and the existence of arsenic in it, might be easily overlooked. On the whole, these salts of copper are seldom used as poisons; although so easy of access, that they are to be purchased without difficulty in any colour-shop. The accidents that arise from them are generally observed among colour-makers and paper-stainers.

Copper in organic liquids.—The oxide of copper is liable to be precipitated by certain organic principles, as albumen, fibrin, and mucous membrane: but some of these organic compounds are easily dissolved by acids, or even by an excess of the solution of cupreous salt. A portion at least of the salt of copper is, therefore, commonly held dissolved. In such cases, there is one peculiar character possessed by these liquids, *i. e.* they have a decidedly *green colour* even when the copper-salt is in a far less than poisonous proportion.

Separation by iron.—We first filter the liquid, and save the insoluble portions for a separate operation. We may use as a trial-test either a needle, zinc with platina, or add to a portion, oxalic acid; the last gives a blueish-white precipitate only when the copper is in moderately large quantity, and the liquid is not very acid. If the needle be not coated with copper in the course of a few hours, it is certain that there is no detectable quantity of the poison present in the liquid. The process by iron will answer notwithstanding the presence of a large quantity of organic matter, and in spite of great dilution; and a very small quantity of a salt of copper may be thus easily discovered in tea, coffee, porter, or gruel, provided we take care to acidulate the liquid slightly with diluted sulphuric acid, before introducing the polished needle. The following is the result of an experiment: One-third of a grain of sulphate of copper was dissolved in water, and mixed with four ounces of thick gruel. Ammonia produced no effect on this liquid; and ferrocyanide of potassium gave only a faint reddish-brown discolouration. Two drops of diluted sulphuric acid were added to it, and a bright needle suspended in it by a thread. In twenty-four hours, the needle was covered with a distinct film of metallic copper. The quantity of metallic salt here present, was less than the 6000th part of the solution. If the needle be rusty, this experiment will fail. The smaller the quantity of copper, the longer the time required for the result to follow. Instead of a needle, a coil of the finest bright iron wire may be used. When the quantity of copper is small, the red colour of the deposited metal is not always perceptible: it appears brown or black, and the deposit may be obscured by its being mixed with some oxide of iron. When we are in doubt about the nature of the deposit, the iron wire should be placed in a small quantity of solution of ammonia and exposed to air. The liquid will soon acquire a blue colour if any metallic copper be present. In this way a grain of sulphate of copper diffused in sixteen ounces of decoction of sarsaparilla and water, was easily detected in a few hours, although the cupreous deposit was not rendered perceptible by its red colour!

Separation by phosphorus.—Orfila long since recommended phosphorus for the purpose of separating metallic copper (Traité des Poisons, i. 508, 3d ed. Paris, 1826,) and it may be sometimes usefully applied. A thin slice of phosphorus is suspended by a thread in the organic liquid suspected to contain copper. If a salt of the metal be present, even in very minute proportion, the phosphorus soon acquires a steel-grey coating (phosphoret of copper.) This continues to increase, the red colour of copper appears, and when the phosphorus is strongly coated, it may be immersed in cold diluted nitric acid. After a short time the copper is dissolved off, and the phosphorus, after being washed in water, may be again immersed. In this way, a solution of nitrate of copper fitted for testing may be procured from the most complex organic liquid, provided any of the cupreous salt be actually dissolved. This is a very delicate process.

Separation as a Sulphuret.—If the copper-salt be present in *large* quantity, any of the trial-tests will indicate it immediately. We now destroy the viscidity of the liquid by diluting it if necessary; and pass into it a current of sulphuretted hydrogen gas in order to precipitate all the copper in the state of sulphuret. The black sulphuret may be collected, washed, dried, and then boiled in equal parts of nitric acid and water for a quarter of an hour. Nitrate and sulphate of copper are produced and dissolved, a fact indicated by the liquid acquiring a rich blue colour: and some sulphur is at the same time separated. This liquid, when filtered, will give the usual reactions with the tests for copper.

Separation by platina.—I have also found the following a very expeditious and simple method of obtaining copper from organic liquids. Having filtered the liquids, let a portion of it be placed in a clean platina capsule or crucible. A few drops of diluted sulphuric acid may be added, and a slip of zinc foil introduced. Wherever the platina is touched by the zinc, metallic copper is deposited; and after having in this way coated the platina capsule, the surplus liquid may be poured off and the capsule well washed out. A small quantity of solution of ammonia poured over the deposit on platina dissolves it and acquires a characteristic blue colour; or a few drops of nitric acid with a small quantity of water, may be used to dissolve the metallic copper; and by evaporating the acid liquid and redissolving the residue in water, a pure solution of nitrate of copper is obtained,—giving the usual reactions with ammonia; and, when the surplus acid is neutralized by an alkali,—with ferrocyanide of potassium and polished iron. Copper, in moderate quantity, may be thus easily separated from milk, gruel, porter, or the most complex organic liquids.

Detection of copper in the tissues.—It may happen, however, that there is no poisonous salt of copper held dissolved in the liquid subjected to analysis,—a fact commonly indicated by the entire want of action on polished iron. The oxide of copper may be intimately combined with some organic principles, or even with the mucous membrane of the stomach itself, and exist only in an *insoluble* form. A piece of this, suspended in a solution of ammonia, is rendered intensely blue if copper be present in moderate quantity. It will then be necessary to cut up these substances, which commonly have a green or blue colour, and boil them for an hour, in water containing one-sixth part of strong nitric acid. The acid liquid should be filtered, and evaporated to dryness; and if much organic matter be present, this should be destroyed by re-digesting it in strong nitric acid, and again evaporating to dryness. Water will now dissolve out any copper as nitrate, which may have been taken up by the nitric acid. If even this process should yield no copper, the organic matter, thoroughly dried, may be incinerated with two parts of black flux in a crucible. The fine particles of carbonaceous ash derived from organic matter often have a shining iridescent red colour, which must not be mistaken for that of copper. By pulverizing this residue, then carefully rubbing it in a mortar with water, and decanting the liquid, minute particles of metallic copper may be obtained, which should be dissolved, in diluted nitric acid, and tested in the usual way. Copper may be also extracted by carbonization with sulphuric acid, or by entirely decomposing the tissue by nitro-muriatic acid.

Normal copper.—It has been objected to the process of calcination, that copper is contained as a natural constituent in most of the organs of the body, and the term *normal copper* has been applied to it. According to Sarzeau, this metal is also present in the incinerated residue of sugar, coffee, madder, wheat flour, and cheese. Blood, milk, and other liquids of the body, in cases where no poisonous salt of copper has been taken, are said to have also yielded it. One chemist made a mixture of eggs, some strong coffee, and bread and butter; he dried and incinerated the mass, and detected copper in the residue! The metal is said to have been found in bread, beef, and mustard. Thus, then, according to this view, copper exists naturally, not only in the organs of the human body, but likewise in some of the most common articles of food. It is, however, very probable that copper may, in these cases, have been introduced accidentally during the analysis, and thus have led to an erroneous inference, especially as it was only found in infinitesimal traces. Dr. Christison could not detect any portion of the metal in the animal fluids; and in some experiments on large quantities of oatmeal and bread, I did not detect the smallest portion of copper, although the tests answered perfectly when a cupreous salt was purposely added in minute proportion. MM. Danger and Flandin have more recently arrived at the same results, *i. e.* that neither copper nor lead enter into the composition of the healthy human body or of the food of man; and that where they are said to have been detected, their presence must be ascribed to their adventitious introduction during the analysis. The question is of some interest in toxicology, for it has been already brought forward as an objection to medical evidence. In August 1843, M. Barse communicated to the Academy of Sciences the results of some analyses made on the bodies of two subjects taken from the hospital of Paris. They had died from ordinary disease. M. Barse states that he detected copper and lead in both subjects. The copper was obtained in the metallic state, and identified by all its characters; the lead was not obtained as a metal, but its presence was indicated by the usual tests. These metals may be detected in the liver, according to M. Barse, 1, by Orfila's process of carbonization; 2, by simple carbonization, incineration of the ash, and afterwards digesting it in nitro-muriatic acid; 3, in carbonizing by sulphuric acid and *incinerating charcoal*, for the mere carbonizing action of sulphuric acid will not of itself suffice to allow of the separation of the metals. In September 1843, M. Rossignon, of Lyons, addressed a note to the Academy of Sciences, on copper as it exists in the organic tissues of many vegetables and animals. M. Rossignon states that he detected copper in all his experiments on the human body: he found it in the blood and muscular fibre of man, in the tissues of many domestic animals (the dog,) and in the common vegetables used as food. The gelatin used as soup at the hospital of St. Louis yielded per cent. 0·03 of pure copper. Common sorrel gave two per cent. of neutral oxalate of copper; chocolate from 0·07 to 0·5 per cent. The bread generally used in Paris gave, in 1000 parts of incinerated residue, from 0·05 to 0·08 of copper (fraudulently introduced as sulphate?) Coffee, chicory, madder, and sugar yielded traces of the metal,—in the latter case mixed with lead. Barley-sugar contains copper: and in the sugar of starch it forms four per cent. by weight, of the carbonized residue. M. Rossignon further states, that by calcining the substances in close vessels, he was enabled to detect appreciable traces of the metal in human semen, in the excrement of the fowl, in the egg, and in the eye of the ox! These results are directly opposed to those obtained by MM. Danger and Flandin, M. Chevallier, M. Chevreul, and others. It is intimated that the failure of these experimentalists in detecting copper, was owing to their not having incinerated the carbon derived from the action of sulphuric acid on organic matter; but this does not sufficiently account for the difference, because, by pursuing the same process with the pulmonary exhalations of animals *poisoned* with its salts, they detected the metal readily, although here it was only found in traces! Besides, when we consider the very positive manner in which it was for a long time stated that arsenic was a normal constituent of the human body, by a higher authority than either M. Barse or M. Rossignon, and that this statement has been since entirely disproved before a committee of the Academy, we may well hesitate to assent to the assertion that copper is a natural constituent of the body. While this sheet was passing through the press, Orfila repeated his experiments on the alleged existence of *normal* copper, before a committee of eminent chemists. The result was, that it required the incineration of *three healthy livers*, and digestion of the ash in nitro-muriatic acid, in order to procure any evidence of the presence of copper! (Journ. de Chim. Août 1847, p. 434.) Any analyst, therefore, who operates upon three dead subjects at once, must be prepared to meet this objection! Practically speaking, it has no force:—1. Because in poisoning by copper, there would be very few cases in which the whole of the chemical evidence rested on an incineration of the viscera:—such a case is very unlikely to occur; for chemical evidence is in general abundantly afforded by an analysis of a portion of the poisoned substance swallowed, or of the contents of the stomach. 2. If the only chemical evidence were that derived from incineration, then this could afford no proof of poisoning, unless that fact were already sufficiently made out by symptoms, post-mortem appearances, and moral circumstances, in which case such infinitesimal proof might be very easily dispensed with. In a case of falsely imputed poisoning, it may be said that the detection of copper in a particular article of food, such as bread, would lead the medical jurist into error, since the discovery of this metal in the bread, might bear out the imputation, and inculpate an innocent person. This hypothesis does not appear probable. The normal copper, said to exist in food, has not been found to form, according to its discoverers, more than the 100,000th part of the food examined:—if the imputation of poisoning were well-founded, and copper were discovered at all, the metal would be in infinitely larger proportion than this, so as to leave no doubt of its actual admixture.

M. Boutigny has pointed out that, in the process of *incineration*, the copper may be concealed and withdrawn from the action of the tests, by the presence of iron in the acid liquid. He has therefore advised that this should be first got rid of by the addition of ammonia.

Copper in the soil of cemeteries.—It is not very probable that a medical jurist would be required to seek for a cupreous poison in a body which had been so long interred as to have its remains intermixed with the soil. But it is not the less necessary to state that according to the researches of M. Walchner, copper, like arsenic, is almost universally found in ferruginous soils, and in most kinds of marls and clays. Wherever the ores of iron exists, there copper will be found: in this way it may be dissolved by water, and percolate through the superficial strata. (See Comptes Rendus, Sept. 21, 1846, 612.) Admitting the truth of this observation, a comparative analysis of the earth of the cemetery, would be required in the very rare case in which the decomposed remains of the dead had become intermixed with the soil. M. Walchner simply digests the earth in muriatic acid, and precipitates the copper from the acid solution by a current of sulphuretted hydrogen gas.

Absorption of Copper.—Copper, like other metallic poisons, is absorbed. It has been discovered in the blood, organs, and secretions, when its salts have been taken. Orfila has found the metal in the lungs, heart, liver, spleen, and kidneys, of animals poisoned by it; but he could discover no traces of it in the blood or urine, although it must undoubtedly exist in the blood. M. Flandin asserts that copper is not found in the heart and kidneys. (Des Poisons, i. 569.) Wibmer, according to Sobernheim, detected copper in the liver of a dog to which he had for several weeks given small doses of the sulphate. Absorbed copper is most easily detected in the liver. About two ounces have been found sufficient for the experiment. MM. Danger and Flandin have recently stated, that copper in cases of poisoning is to be detected more readily in the bronchial secretion than in the urine. (Annals d'Hyg., 1843, 452.)

QUANTITATIVE ANALYSIS.—This is best determined by converting the salt of copper to the state of black oxide, every 100 parts of which are equal to 312 of crystallized sulphate, and 392 of crystallized nitrate. If the cupreous salt be precipitated as sulphuret, this may be transformed to black oxide by digestion in nitric acid, and subsequent incineration.

COPPER IN ARTICLES OF FOOD.

The medico-legal history of poisoning by copper, would be incomplete without some remarks on the action of certain articles of food on this metal when used for culinary purposes. This is a not unfrequent form of accidental poisoning. The symptoms rarely appear until after the lapse of three or four hours:—these are commonly nausea with colicky pains and cramps in the limbs. Several fatal cases are on record. (Galtier, i. 626.)

It results from the experiments of Falconer and others, that metallic copper undergoes no change by contact with *water*, unless air be present; when a hydrated carbonate will be formed mixed with peroxide. If the water contain any acid, such as vinegar, or common salt,—or there be oily or fatty matter in contact with the metal, then the copper is more rapidly oxidized, and the liquid or fat acquires a green colour. If the copper vessel be kept perfectly clean, and the food prepared in it be allowed to cool in other vessels, there is not much risk of its acquiring a poisonous impregnation: nevertheless no acid, saline, fatty, or oily liquid should be prepared as an article of food in a copper vessel. (See Ann. d'Hyg. 1832, i. 102.) Under the influence of heat and air, a portion of copper becomes dissolved, and the oily or other liquid acquires a green colour. The preparation of fruits, such as preserves, in copper vessels, is necessarily attended with some risk; for on cooling, a green crust is apt to form on the copper, just above the surface where the air and acid liquid meet. Some substances appear to be but little liable to this impregnation:—thus, coffee, beer, milk, or tea has been boiled for two hours together, in a clean copper vessel, without any portion of the metal being taken up by either of the liquids. (See Falconer on the Poison of Copper, 65, London, 1774; also Orfila, i. 611.) Accidents of this kind are usually prevented by lining the copper vessel with tin; but in very large boilers this plan is not always adopted—cleanliness alone is trusted to, and this is a sufficient preventive when properly observed.

The fatal effects resulting from the impregnation of *acid liquids* with copper, are established by the following accident, which occurred a few years since. A servant girl at a farm house put a copper vessel into a tub, containing the wash with which the pigs were fed. This is said to be a common practice in many parts of the country, as the acidity of the liquid serves to cleanse the copper vessel. A number of pigs were fed with this wash, and six of them died;—their bodies were examined, and the stomachs were found inflamed. Owing to the ignorance which prevails on these matters, soup and other articles of food, such as acid wine, beer, or cider, are often improperly kept in copper vessels, and thus become poisoned. Dupuytren relates that a whole family was poisoned by eating cray-fish, cooked and allowed to cool in a copper vessel, to which vinegar had been added. Three persons died.

In April 1888, I was required to examine the following case. In an extensive Poor-law union, a number of the paupers had been seized with diarrhœa and dysentery, and several of them died. There was no apparent cause for this sickness and mortality; and it was suspected that the soup, which was daily prepared in large copper boilers, might have become impregnated with the metal, and have given rise to the symptoms, although these were scarcely indicative of irritant poisoning. I ascertained that the copper vessels were cleaned out daily, that the soup was made with salt and vegetables, but was poured into other vessels to become cool. The soup was given only once a week; but the gruel, which was also suspected, was given daily to the paupers. Four ounces of the soup, clarified by standing, gave no trace of copper by the iron-test; and the liquid was wholly unaffected by a current of sulphuretted hydrogen gas. Eight ounces were evaporated to dryness, and calcined with flux;—an abundant ash resulted, presenting iridescent colours, many portions having a bright *coppery* lustre. The ash was treated with water, and the heavy residue digested with diluted nitric acid. The filtered liquid, when neutralized, gave no sign of the existence of copper with any of the tests:—iron was present, as it is in the incinerated residue of most vegetables. This experiment, while it showed the absence of copper as a poison, also appears to prove that articles of food do not always contain it as a natural constituent. The result of the analysis of the gruel, was equally negative. The bread was also examined, without any noxious irritant substance being discovered in it. It was therefore evident that the symptoms could not have been due to irritant poison.

One of the most satisfactory accounts of *slow poisoning* by copper, from want of cleanliness in the use of culinary utensils, has been lately published by Dr. T. Moore. It shows that without great circumspection, a medical man may be completely deceived respecting the origin of a malady affecting many persons simultaneously (p. 50, ante.) On the return of the Indian Coolie emigrants from Guiana to Calcutta, a kind of acute idiopathic dysentery made its appearance in the ship, and it was at first referred to bad water, change of climate, and other causes. Dr. Moore examined the copper-plates on which the fish, rice, and ghee (butter,) eaten by the natives, was cooked, and found the surface quoted with a green composition which, when scraped off and examined, proved to be a mixture of muriate and sulphate (?) of copper. The cause of the disease was then apparent. A few hours after taking the meal, the patients complained of violent pains and cramps in the stomach and lower bowels, and there was constant vomiting of greenish and yellowish-green bile. When this was not ejected from the stomach, their sufferings from dry retching were most severe: and the feeling of constriction in the lower part of the chest and along the course of the œsophagus still more distressing. Every twenty minutes there was an attempt to evacuate the bowels, but no feculent matter was discharged: blood in small quantities, slimy mucous stools, tinged with blood, shreds of lymph, and frothy ash-coloured secretions, were passed from the rectum without affording to the patients, the slightest relief. Pressure over the abdomen, especially in the epigastrium, and in one, on the arch of the colon, caused pungent pain. There were griping pains in the loins and sacrum, at the navel, and in the iliac region, with tenesmus and a burning sensation at the sphincter ani. In the commencement of the attack, there was acute fever, pungent heat of the skin, headach, urgent thirst, loss of appetite, prostration of strength, furred and clammy tongue, foul taste in the mouth, with a rapid, small and wiry pulse. In the more severe cases there was great depression of the vital powers, the pulse exceedingly rapid and weak, the skin cold, extremities benumbed; the secretion of urine was in a few instances suppressed, in others the urine was retained in the bladder. The symptoms in most instances subsided in eight or ten days under the free use of emetics and castor oil; in others a long time elapsed before the mucous discharges from the alimentary canal and the tenesmus abated,—the disease assuming all the characters of chronic dysentery. One man was subsequently attacked with symptoms of chronic poisoning in an aggravated form, from neglect in the use of a copper-vessel, and sank under the attack. On a post-mortem examination, the mucous membrane of the lower part of the œsophagus, and that of the stomach between the two orifices, was the seat of extensive and deep-seated inflammation. The shades of red varied from a bright vermilion or scarlet to a deep red or violet colour. The patches of a dark red or brownish colour were comparatively small and circumscribed, situated in general beneath the mucous membrane of the under surface of the stomach. The membrane in these situations was softened, pulpy but not excoriated, and free from the appearance of having sloughed. At the pylorus the membrane was intensely inflamed, glistening, and tumid from a quantity of serous fluid exuded beneath the submucous cellular tissue. The mucous membrane of the duodenum and small intestines was also inflamed in irregular patches; and there were traces of inflammation in the large intestines, including the rectum. Eight ounces of a saffron-coloured fluid were found in the peritoneal cavity, and on the peritoneal surface of the intestines there were numerous minute spots of inflammatory redness. There was no effusion of lymph or other sign of peritoneal inflammation. (Lancet, April 11, 1846, 414.) Dr. Moore considers that the attacks of cholera and acute or chronic dysentery, under which Europeans arriving in the East Indies so frequently suffer, may be in many cases due to the general employment of copper-utensils for culinary purposes, and from the want of cleanliness on the part of the native cooks, who use butter, salt, and acids, without removing the cupreous incrustation which is formed on the surface or in the rims of the vessel. Hot butter or lard, like hot oil, readily dissolves copper, forming fatty salts of which oxide of copper is the base.

In the making of preserved *fruits* and vegetable *pickles*, the salts of copper (blue vitriol) are sometimes used for the purpose of giving a rich green colour. Many of the green pickles, sold in shops, are thus impregnated with the vegetable salts of this metal, to which they owe their bright grass-green colour. If the fruit or pickle be placed in a solution of ammonia, and copper be present, the substance is speedily turned blue. The iron test is, however, more delicate. A needle immersed in the pickle, or plunged into the solid, will be speedily coated with copper. The quantity of copper contained in such articles may not be sufficient to produce fatal effects; but serious symptoms of gastric irritation are sometimes produced, and in very young subjects, these may assume an alarming character. (See Falconer, 87.) A short time since preserved gooseberries were sent to me for examination, as it was suspected from circumstances that they were contaminated with copper. The liquid in which they were preserved was of a pale yellow colour, and had an acid reaction.

Ammonia gave with it a dark greenish tinge; ferrocyanide of potassium a rich claret-red precipitate; and hydrosulphuret of ammonia a deep brown. A needle plunged into it, was coated with copper in about five or six minutes. The galvanic test applied in the usual way, failed to indicate the presence of the metal. I therefore modified it by plunging into the liquid a slip of platina, having a coil of zinc twisted round it:—copper was then deposited on the platina in the course of a short time. The liquid gave a precipitate with nitrate of barytes, insoluble in nitric acid. These facts proved that the liquid was pretty strongly impregnated with sulphate of copper!—and the analysis shows that ammonia is decidedly inferior to the other tests, the blue colour having been concealed and changed to green by the yellow colour of the liquid. The test upon which reliance may always be placed is polished iron; and this, while it is the least open to objection, happens at the same time to be very delicate in its reaction, and less affected than the others by the presence of organic matter. Colic and vomiting have been produced by the use of vinegar which had been placed in copper-utensils; and copper stop-cocks to vessels containing acid liquids, may thus give rise to symptoms of poisoning.

Bread poisoned by copper.—A few years since a fraudulent practice existed on the continent, of mixing sulphate of copper with the dough of *bread*, in in order, as it was said, to accelerate the panary fermentation. The quantity of cupreous salt used was small, but still it was a noxious adulteration. (Ann. d'Hyg., 1830, 342; 1831, 338; 1840, 2, 123.) According to some experimentalists, *bread* always contains traces of copper, which is supposed to form a normal constituten of corn (ante, p. 382.) Reasons have been already given for rejecting this opinion. Copper may have been detected in this article of food, but this does not prove that it is a normal constituent. It may have been introduced accidentally during the making of the bread, as where copper-utensils have been used for this purpose. Thus it may be found in bread, and not in the flour from which the bread was made, or in the flour and not in the corn. MM. Theuleu and Servan having found copper in a specimen of bread, ascertained by further examination that copper cylinders had been used in grinding the corn. A small quantity of oxide falling from these would at once account for the contamination irrespective of fraud. (See Orfila, i. 651; Galtier, i. 607.) M. Girardin has more recently pointed out another source from which the copper may be derived. Seed corn is sometimes dressed (to destroy the spores of fungi) with a mixture of common salt and sulphate of copper. He has analysed the grain grown from seed thus treated, and has distinctly recognised in it traces of copper. (Annuaire de Chimie, 1846, 686.) Paper used for filtration sometimes contains traces of copper. Alum is used in bread as well as salt, and small quantities of copper sometimes exist in these substances. These facts tend to show that those who assert that copper is a normal constituent of most kinds of food, have not been sufficiently careful in their inquiries. Bread containing even traces of sulphate of copper, acquires a red tint when moistened with ferrocyanide of potassium. If the proportion be greater, its taste and colour are affected: it may have a blueish-green colour. Copper may be obtained from it by the process of incineration.

Accidental poisoning by copper has occurred from the use of what is called *German silver*, but which should rather be called *white brass*, as it is an alloy of copper and zinc with nickel. Some specimens of this alloy contain fifty per cent. by weight of copper. The following case of poisoning occurred in Paris in 1838. A lady, after having had eels for dinner, was awakened in the night by intense headache, followed by nausea, vomiting and colic. These symptoms were removed under proper treatment. Her physician ascertained that the eels had been cooked with butter and vinegar in an earthenware vessel, and he found that the metal spoon, which was of German silver, presented on different parts greenish-coloured spots. Chemical analysis showed that a poisonous salt of copper had been thus accidentally produced:—a fact proved by polishing the spoon, and then placing it in a hot mixture of bread, butter, and vinegar. Half an hour after the mixture had cooled, green spots were perceived on it; and in twelve hours the spoon was quite green, as well as the butter in contact with it. It has been proposed to prevent this chemical action on copper and its alloys by electro-plating them with silver; but Mr. Warington has found that the silver is deposited unequally like a spongy mass, thus allowing the acid liquid to penetrate through it. A galvanic action is thereby set up, which increases the chemical changes.

Action of water on copper.—Water does not appear to have any marked action on this metal when the surface is clean. On exposing clean metallic copper in contact with *distilled* water and air for a period of forty-three days, the water was clear, without colour, taste, or smell, and the copper bright and unchanged as when first immersed. The tests for copper showed that there was none of the metal dissolved. On evaporating three ounces to dryness, however, a very slight green sediment (less than one-eighth of a grain) was procured, which, on analysis, was found to be carbonate of copper. When *river water* was substituted, similar results were obtained, but the carbonate of copper left on evaporation, was in rather larger quantity. This may have arisen from the presence of bicarbonate of lime. The contact of water with clean copper-vessels, is therefore not likely to lead to the production of a sufficient quantity of poisonous salt to affect health. When the copper is corroded or dirty, the poisonous crust may become mechanically diffused through the water, and give rise to chronic poisoning.

CHAPTER XXIX.

ANTIMONIAL VAPOURS. TARTARIZED ANTIMONY—LARGE DOSES NOT INJURIOUS—ITS EFFECTS ON CHILDREN—SYMPTOMS—FATAL DOSE—RECOVERY FROM LARGE DOSES—EXTERNAL APPLICATION—POST-MORTEM APPEARANCES—TREATMENT—CHEMICAL ANALYSIS—DELICACY OF THE TESTS. ANTIMONY IN ORGANIC LIQUIDS—DETECTION IN THE TISSUES. ARSENIC IN ANTIMONIAL PREPARATIONS. ANTIMONY IN SOLIDS—QUANTITATIVE ANALYSIS. CASES OF POISONING BY CHLORIDE OF ANTIMONY—ANALYSIS. OTHER ANTIMONIAL PREPARATIONS. POISONING BY SULPHATE OF ZINC—ANALYSIS—IN ORGANIC MIXTURES—ZINC IN THE TISSUES—EFFECT OF WATER ON ZINC—CARBONATE OF ZINC—OTHER PREPARATIONS OF THIS METAL

General remarks.—Metallic Antimony is not regarded as a poison, but when respired in the state of *vapour*, it is stated to have produced serious symptoms. A case of poisoning by the vapours of antimony is reported in the Edinburgh Medical and Surgical Journal (lv. 265.) Orfila suggests that the effects said to be produced by this metal in vapour may be ascribed to arsenic, which is present in most specimens of crude antimony as it is used in manufactures. (Toxicol. i. 504.) Of the antimonial compounds, there are two which may be specially considered, namely, Tartar Emetic and Chloride of Antimony.

TARTARIZED ANTIMONY. TARTAR EMETIC. STIBIATED TARTAR.

This substance, which is seen in the form of a white powder, or in crystals, is by no means so poisonous as it is often described to be. Forty grains have been given to an adult in twenty-four hours without causing serious mischief. Professor Forget of Strasburg has related the case of a robust man, aged forty, who, while labouring under acute rheumatism, took tartar emetic, first in the dose of eight grains, increasing it gradually to sixty, and then to seventy-two grains. He took this quantity without any disorder of the intestinal canal, or any other bad symptom. In the space of ten days, the man took without inconvienence *three drachms* of tartar emetic. (Med. Gaz. xxiv. 126; see also a case in Orfila, ii. 743.) Nevertheless, other facts show, that this substance, in doses of from half an ounce to one ounce, or even less, must be regarded as an irritant poison; and one reason why the symptoms are often so slight from comparatively large doses, is owing to its possessing such violent emetic properties. This leads to the early expulsion of the greater part of the poison from the stomach. A case is related by Dr. Lambert, where only *four grains* of tartar emetic gave rise to violent pain in the abdomen, vomiting and purging. The individual then fell into strong convulsions, which lasted half an hour. He became speechless, no pulse could be perceived, and the skin was quite cold;—in short, it was supposed he was dead. Stimulating frictions and cataplasms were employed, and he slowly recovered in about fourteen days. (Casper's Wochenschrift, xiii. 1241.)

It would appear from the observations of the late Mr. Goodlad of Manchester, and Mr. Noble, that tartarized antimony, even in small doses, is liable to act as a poison on the young. Mr. Wilton records four cases in which prostration and collapse followed the administration of ordinary doses of tartar emetic to young children. Two of them were fatal. It should therefore be administered with great caution.

A case was referred to me in March 1847, in which it was suspected that a child labouring under disease of the lungs, had been killed by an over dose of tartarized antimony. The child took two doses of an antimonial mixture, and died twenty-four hours after the last dose. There was no vomiting, purging, or any other symptom, excepting sudden access of pain, to lead to the suspicion that the medicine had acted as a poison. The determination of the quantity present in each dose became of course very material, in order that the medical practitioner might escape a charge of manslaughter. By a quantitative analysis to be subsequently described, the proportion of tartarized antimony in each dose was only 0·29 grains: hence the deceased had taken in the two doses, but little more than *half a grain*. An opinion was therefore given, that the child had not died from the effects of the medicine, since it would not have been just to have drawn such an inference from the occurrence of a few exceptional instances, such as those reported by Mr. Noble and Mr. Wilton. From a case published in the London Medical Gazette, it appears probable that the life of a child was destroyed by a dose of fifteen grains given by mistake for another powder. (xvi. 521; see also xl. 351.)

Tartar emetic appears to act more as an irritant than as a corrosive; but the symptoms which it produces, like those of all corrosive poisons, are generally immediate. In several instances this substance has proved fatal in England. In one, a man, aged twenty-four was killed by a dose of three drachms taken by mistake: (Traill, 114) and one or two fatal instances are reported by Orfila to have occurred in France. Our knowledge of its effects as a poison on man, is chiefly derived from the cases related by Orfila. In 1837, a trial took place on the Norfolk circuit, for the administration of this substance with intent to murder; but there was a total want of proof: the tartar emetic was given to a child medicinally by the prisoner, an ignorant woman, without there being apparently any intention on her part to destroy it.

This substance is used in medicine both externally and internally. TARTAR EMETIC SOLUTION, OR VINUM ANT. POT. TART., contains one grain in half an ounce.

It is exhibited in doses of fifteen drops to one drachm. Tartar-emetic ointment contains one-fifth of its weight of this substance.

SYMPTOMS.

A strong metallic taste is perceived in the mouth during the act of swallowing. There is violent burning pain in the epigastric region, followed by nausea, vomiting, profuse diarrhœa and syncope. The pulse is small and rapid, sometimes imperceptible; the skin cold, and covered with a clammy perspiration; and the respiration painful. Death is preceded by vertigo, insensibility, great prostration of strength, and violent spasms of the muscles of the extremities. Among the symptoms there has been observed great constriction in the throat, with difficulty of swallowing. The *quantity* actually required to destroy life is unknown. It will probably depend much on whether active vomiting and purging have been excited or not; for these symptoms have not been present in all cases. Doses of twenty, twenty-seven, and even sixty grains have been taken without destroying life, although alarming symptoms of irritation followed. In one case related by Orfila, a man aged fifty, took forty grains of tartar emetic and died in about four days. This was the only one out of five cases of poisoning by this substance which proved fatal. (Orfila, i. 447.) Dr. Beck mentions a case in which fifteen grains of tartar emetic, in solution, killed a child a few weeks old: vomiting and purging ensued, followed by convulsions and death. In two cases observed by Mr. Hartley, which will be presently described, *ten grains* killed each child in a few hours. This, I believe, is the *smallest fatal dose* on record. In a case recently reported by Mr. Freer of Stourbridge, a man ætat. 28, swallowed *two drachms* of tartar emetic by mistake for Epsom salts, and recovered from its effects. An hour after the poison had been taken, he was found in the following state:—his pulse imperceptible; tongue dry and red; countenance cold and livid, bathed with clammy perspiration, and indicative of great suffering; violent pain at the epigastrium and over the whole of the abdomen, with constant spasmodic contraction of all the muscles, particularly of the abdomen and upper extremities. The fingers were firmly contracted, and the muscles quite rigid. He vomited only once, about *half an hour* after he had swallowed the poison, and after this he had constant involuntary aqueous stools. An emetic of mustard and salt was given to him, and this produced violent vomiting of bilious matter. Green tea, brandy, and decoction of oak-bark were freely given. The cramps, vomitings, and aqueous stools continued for six hours. The symptoms then became mitigated, and he gradually recovered, suffering chiefly from profuse night perspirations. (See Lancet, May 22, 1847, 585.) This case is remarkable for the anomalous character of the symptoms, as in the absence of active vomiting, an emetic was actually required to be given,—also from the recovery of the individual after a very large dose of the poison.

External application.—Tartar emetic is said to have produced symptoms of irritant poisoning when applied externally to the skin, in the form of ointment as a counter-irritant. In a case where the skin was but little affected by the use of this ointment, nausea and sickness were produced, which disappeared when the use of the ointment was discontinued. Although it is very extensively used by medical practitioners, we never hear of cases of poisoning by it under these circumstances. Dr. Griffith, of Philadelphia, states that, thus applied, it has produced violent salivation. (Am. Jour. Med. Sci., II. 233.)

POST-MORTEM APPEARANCES.

In Orfila's case above mentioned, the mucous membrane of the stomach and duodenum was reddened and covered with a slightly adhering layer of mucus. In a man who had taken forty grains of tartar emetic during a period of five days, and who then died from an attack of apoplexy,—the stomach was found much reddened and inflamed in irregular patches, the redness passing into a violet tint; but there was no ulceration of the mucous membrane,—the duodenum was in a somewhat similar state, and the small intestines were but slightly inflamed. The following cases reported by Mr. Hartley, show the nature of the post-mortem appearances likely to be found in the body. Two children, a boy aged five years, and a girl aged three years, each swallowed a powder containing *ten grains* of tartar emetic mixed with a little sugar. It was stated that, in twenty minutes after taking the powders they were seized with violent vomiting and purging, and great prostration of strength, followed by convulsions and tetanic spasms: there was also great thirst. The boy died in eight hours, and the girl in twelve or thirteen hours after swallowing the dose. The bodies were inspected between four and five days after death. In that of the boy there was effusion of serum in the right pleura; the lower lobe of the right lung posteriorly was redder than natural, and the peritoneum was injected from recent inflammation. The mucous membrane of the duodenum was inflamed, and covered with a whitish-yellow viscid secretion; this was observed throughout the intestinal canal, though the colour was of a deeper yellow in the colon and rectum: there was no ulceration. The peritoneal coat of the stomach was inflamed. The mucous membrane of this organ was much inflamed, especially about the larger curvature and at the cardiac orifice: there was no ulceration. The contents (about two ounces and a half of a dark grumous fluid, having a slightly acid reaction,) were very adherent to it; and in one place there was a patch of lymph. The tests used did not indicate the presence of antimony. With regard to other appearances, the tongue was covered with a white fur, and appeared soddened; the fauces were not inflamed; the trachea and œsophagus had a natural appearance. On opening the cranium, the dura mater was found very vascular; the longitudinal sinus contained a coagulum of lymph, but very little blood. The vessels of the surface of the brain were very much injected with dark blood, the whole surface having a deep purple appearance. Every portion of the brain, when cut, presented many bloody points. The cerebellum and medulla oblongata were also extremely vascular; there was no effusion in the ventricles, or at the base of the brain. In the body of the girl, the morbid appearances were similar; there were also patches resembling the eruption of scarlatina on the arms, legs, and neck. The arachnoid membrane was more opaque than usual; and on the mucous membrane of the stomach, where the inflammation was greatest, were two or three white spots, each about the size of a split pea, which appeared to be the commencement of ulceration. (Lancet, April 25, 1846, 460.) In animals poisoned by this substance, it is common to find general inflammation of the alimentary canal.

TREATMENT.

This consists in promoting vomiting by the free administration of warm water, milk, or other diluents. The stomach-pump may also be used. Any vegetable infusion containing tannin, such as strong tea, decoction of oak-bark, or Peruvian bark, may be given. This principle combines with oxide of antimony, to form a compound insoluble in water; and if attended with no other benefit, it at least suspends the operation of the poison. This tannate of antimony is said to be inert; it is easily dissolved by some vegetable acids albumen, or the paper be allowed to become dry, before it is exposed to sulphuretted hydrogen, the stain is yellow, thus resembling that of arsenic. So Sobernheim has observed a similar effect when tartar emetic is mixed with solution of gum. In analyzing the contents of the stomach, we might therefore be erroneously led to suspect the presence of arsenic, since tartar emetic is frequently given as a medicine. Having ascertained that antimony is present, the liquid is strongly acidulated with tartaric acid, and a current of sulphuretted hydrogen gas is passed into it until there is no further effect. The sulphuret is collected, washed, and dried. If it be the sulphuret of antimony, it will have an orange-red or brown colour, and will, when dried, be dissolved by a small quantity of boiling muriatic acid (forming sesquichloride of antimony) with evolution of sulphuretted hydrogen. The boiling should be continued for several minutes. On adding this solution to a large quantity of water, a dense white precipitate of oxychloride of antimony (powder of Algaroth or Algarotti, *Mercurius Vitæ*) will fall down. This is characteristic of antimony. If it be objected that nitrate of *bismuth* undergoes a similar change when dropped into water, hydrosulphuret of ammonia will easily enable us to distinguish the two metals; the antimonial precipitate is turned of an orange-red by that solution, while the bismuthic precipitate is turned of a deep black. Besides, the white precipitate from antimony is known from that of bismuth by its more ready solubility in tartaric acid. Dr. Turner recommended that the precipitated sulphuret of antimony should be reduced by heating it in a current of hydrogen; but there are some objections to this. Dr. Turner himself found that organic matter became precipitated with the sulphuret, and interfered with the metallic appearance after its reduction; and even supposing the metal to be obtained, it will require to be identified by certain chemical processes. The production of the chloride from the sulphuret, with its peculiar properties, is more expeditious and quite satisfactory.

Detection of absorbed antimony in the tissues.—If these processes fail, antimony may still be discovered in the solid tissues of the body. For this purpose, Orfila recommends that the viscera should be thoroughly dried and added gradually to boiling nitric acid, until dissolved. Evaporate to dryness and carbonize. Boil the carbonaceous residue in muriatic acid with a little nitric acid. This converts the antimony to chloride,—a portion of which may be introduced into Marsh's apparatus, and tried for antimonial sublimates. If these be obtained, they may be tested in the way described. (See ante, page 283; also, APPENDIX.) By this process, Orfila has succeeded in detecting antimony in the urine, liver, and other viscera,—a clear proof that it is absorbed. He failed to discover it in the blood, or in any animal fluid except the urine. (Annales d'Hyg. 1840, 474.)

During life this metal may be detected in the urinary secretion of those who are taking medicinally antimonial preparations, even for so long a period as twenty-four days after its administration. The plan recommended by MM. Millon and Laveran for its detection, is to add to ten parts of urine one part of pure and fuming muriatic acid, and to stir the mixture with a polished tin rod. If antimony be in large quantity, the tin will be blackened in the course of a few hours; if in small proportion, several days may be required for the deposit. Warmth accelerates the precipitation. A fresh piece of tin must be used in every experiment. These experimentalists have made the singular discovery with respect to antimony, that there are intermissions in its elimination from the body, and that these are great in proportion to the length of time which has elapsed since its administration. (Comptes Rendus, 1845, ii. 638.)

According to MM. Danger and Flandin, antimony will be found most abundantly in the liver after death.

A medical jurist must remember that the discovery of tartar emetic in the contents of a stomach is by no means a proof of its having been taken or administered as a poison; since it is frequently prescribed as a medicine, and often taken as such by persons of their own accord. We could only infer that it existed as a poison, or had caused death, when the quantity present was very large, and there were corresponding appearances of irritation in the alimentary canal. Still less would the discovery of it in a mixture, unless in very large proportion, be evidence of an intent to poison.

Arsenic as an adulteration in antimonial preparations.—It is important, perhaps, in a medico-legal view, to state that arsenic has been discovered by Serullas, to exist in the common sulphuret of antimony, in the metal, and in the preparation called kermes. In the common sulphuret, it has been found in the proportion of from two to five per cent. It has been supposed that pharmaceutical preparations of antimony may be thus contaminated with arsenic; but it does not appear that tartar emetic, when well crystallized, contains any traces of this poison;—the mother liquor contains it, and sometimes the last crops of crystals which are obtained from the solution, may hold a portion of arsenic.

If any antimonial preparation has been exhibited medicinally to a person alleged to have died from arsenic, and arsenic is discovered in the body, the medical witness must be prepared for this objection to chemical evidence. Two persons were recently tried in France for a double murder by poisoning with arsenic; and arsenic and antimony were detected in the exhumed bodies. The presence of antimony was accounted for by the fact that each of the deceased, had taken before death an antimonial quack-medicine. An objection was made to the evidence, that the arsenic might have been mixed with the antimony as an ordinary adulteration in the medicine, none of which could be procured. This view, however, was immediately set aside by the fact that the arsenic was in .very large, and the antimony in very small proportion. It is obvious that such an objection could only hold *cæteris paribus* when the arsenic was in very minute quantity. (Gaz. Méd. Janvier 1846.) This question might easily arise in England under the exhibition of James's powder, antimonial wine, or even of tartarized antimony, given as an emetic to remove the poison.

A clear distinction must be drawn by the witness between the antimonial and arsenical deposits obtained by Marsh's process, in order that the objection may be answered. The detection of minute traces of arsenic in antimony is simple enough, but the detection of minute traces of *antimony* in arsenic, requires more careful manipulation. In a metallic deposit obtained by the combustion of hydrogen, we are certain of having one or the other, or it may be both of these metals. The deposit is heated with a few drops of nitro-muriatic acid, and evaporated to dryness. The residue is digested with a small quantity of distilled water to remove any arsenic acid, and then warmed with a few drops of concentrated muriatic acid. Chloride of antimony is produced, which, if in sufficient quantity, and too much muriatic acid be not present, gives a milky-white precipitate when added to distilled water: and the white subchloride of antimony thus formed, acquires an orange-yellow colour from a current of sulphuretted hydrogen gas, or by the addition of a few drops of hydrosulphuret of ammonia. If no white precipitate be produced on adding the muriatic acid solution (not too acid) to water, there is no certain evidence that antimony is present. (See ante, page 283.)

Further, the antimonial stain is known by its entire solubility in hydrosulphuret of ammonia—the arsenical by its perfect solubility in the vapour of phosphorus. For further details on the distinction between minute films of these two metals, see Appendix, *Deposits of arsenic and antimony.*

In organic solids.—Supposing that there is no antimony in solution, we take the solid substance left on the filter,—the mucus of the stomach or other matters, and boil them in water strongly acidulated with tartaric acid. The insoluble compounds of oxide of antimony are immediately dissolved by this acid. We now filter and pass into the liquid a current of sulphuretted hydrogen gas; sulphuret of antimony is precipitated if any of the poison be present, the vegetable acid not interfering with the action of the gas.

QUANTITATIVE ANALYSIS.—The quantity of tartar emetic present in a liquid, may be determined by the weight of the washed and dried sulphuret of antimony: one hundred parts of the dried sulphuret (sesquisulphuret) by weight, are equal to 202·78 parts of crystallized tartarized antimony.

CHLORIDE OF ANTIMONY. SESQUICHLORIDE OR BUTTER OF ANTIMONY.

This is a highly corrosive liquid, varying from a light yellow to a dark red colour:—in the latter state containing generally a large quantity of iron. It is a powerful poison, but it is not often taken as such. Orfila mentions only one, and that a doubtful instance, which occurred nearly two hundred years ago. I have the accounts of three cases of recent occurrence, in two of which recovery took place, while the other was fatal.

SYMPTOMS AND APPEARANCES.

The following case was communicated to me by Mr. Henry Pearson. In 1836, a boy, aged 12, swallowed by mistake for ginger-beer, four or five drachms of a solution of butter of antimony. In half an hour he was seized with vomiting, which continued at intervals for two hours. There was faintness with general weakness, and great prostration of strength. Remedial means were adopted, and the next day the chief symptoms were heat and uneasiness in the mouth and throat, with pain in swallowing. There were numerous abrasions on the mucous membrane of the mouth and fauces; and there was slight fever, from which he quite recovered in about eight days.

The second case occurred to Mr. Houghton, of Dudley, in 1841. In this instance, about a table-spoonful of the chloride of antimony was given, by mistake for antimonial wine, to a boy aged ten. Immediately on drinking it, the boy seemed choked:—his features were set, and he was unable to speak for some minutes. He vomited freely,—gruel was given to him, which was rejected: he complained of great pain in his throat. Medical assistance was sent for, and about two hours after swallowing the poison, the child laboured under the following symptoms. The features were pale and collapsed, the eyes sunk,—the pupils dilated and inactive,—the skin cold,—the mouth filled with a thick tenacious transparent mucus,—nausea, vomiting,—pulse 80 and small, and breathing heavy. He was in a kind of stupor, from which he could, however, be roused to answer questions rationally. He felt a severe burning pain in the throat, extending to the stomach,—increased by deglutition. Under active medical treatment, these alarming symptoms were removed: on the following day it was observed, that there were patches of a bright scarlet colour in the throat, with difficulty of swallowing. In the course of a few days the boy recovered.

Mr. Bancks, of Stourbridge, has more recently reported the following remarkable case of recovery from a dose of the chloride of antimony. On the 16th November, 1846, A. B., a little boy aged seven years, swallowed two drachms of chloride of antimony, sent in mistake by a druggist, who immediately discovered his error and applied for medical assistance. There was excoriation of the mouth and fauces; the skin was cold and clammy; pulse small and accelerated; burning pain in the epigastrium; tumefaction of the bowels, and incessant vomiting. Magnesia diffused in water was freely given to neutralize the acid. At 3, P. M., the decoction of yellow cinchona and strong tea were given, and continued at intervals, until 8 P. M., when there appeared much less pain in the epigastrium, although a great deal of febrile action was going on. The plan of treatment now adopted was antiphlogistic. Tea with mild diluents, in large quantities, and an enema, were ordered. The boy gradually recovered, and on the 20th was out of danger. For the next few days he continued to improve, and was soon in perfect health again. It is worthy of remark that the child had taken no food on the morning he swallowed the poison,—a circumstance much against the chance of recovery. (Prov. Med. Journ. Dec. 23, 1846.)

The only fatal case which I have met with, was communicated to me by Mr. Mann, of Bartholomew Close. An army surgeon swallowed, for the purpose of suicide, from two to three ounces by measure, of chloride of antimony. About an hour afterwards, he was seen by Mr. Mann. There was entire prostration of strength, with coldness of skin, and incessant attempts to vomit. The most excruciating griping pains were felt in the abdomen; and there was a frequent desire to evacuate the bowels, but nothing was passed. In the course of a few hours reaction took place, the pain subsided, and the pulse rose to 120. There was now a strong disposition to sleep, so that he appeared as if labouring under the effects of a narcotic poison. In this state he continued until he died,—ten hours and a half after he had swallowed the poison. On inspection, the interior of the alimentary canal, from the mouth downwards to the jejunum, presented a black appearance, as if the parts had been charred. In general, there was no mucous membrane remaining, either on the stomach or elsewhere; only a flocculent substance, which could be easily scraped off with the back of the scalpel, leaving the submucous tissues and the peritoneal coat. All these parts were so soft that they were easily torn with the fingers.

TREATMENT.—The free exhibition of magnesia as well as substances containing tannin. See TARTAR EMETIC. (Page 392.)

CHEMICAL ANALYSIS.

If any portion of the chloride be left in the vessel, it may be tested by adding a few drops to a large quantity of water, when the whitish-yellow oxychloride of antimony will be precipitated: the supernatant liquid will contain muriatic acid, which may be detected by nitrate of silver. It has been already observed, that the only objection to this mode of testing, is, that the salts of *bismuth* are also decomposed by water; but the precipitate in this case is insoluble in tartaric acid, and is blackened by hydrosulphuret of ammonia; while in the case of antimony, it is soluble in that acid, and is changed to an orange-red by the hydrosulphuret. If the chloride contain much iron, it will be proper to separate the white precipitate, and wash it thoroughly with water, before adding the hydrosulphuret, or the sulphuret of iron formed, will conceal the orange-red colour. A piece of copper, when heated in a solution of chloride of antimony, is immediately coated with a layer of this metal of a grey colour, like arsenic. Solutions of tartar emetic and of chloride of antimony are very differently affected by tests. Nitric acid precipitates the former, but not the latter. Ferrocyanide of potassium has no effect on a solution of tartar emetic, but it precipitates the chloride of antimony of a yellow-white; or if much iron be present, Prussian blue is abundantly thrown down. The chloride, as a corrosive, combines with the animal tissues. The antimony may be separated in such cases by boiling them in muriatic or nitro-muriatic acid. In this way, the organic matter will be decomposed and entirely destroyed—the antimony being recovered on evaporation to dryness.

The Sulphurets and Oxides, including the Glass of Antimony, or vitrefied Oxysulphuret and Kermes, may be regarded as irritant poisons, although but little is known concerning their action on the human subject. A case of attempted poisoning by the administration of the Glass of Antimony in soup, is reported by Chaussier. There appears to have been no doubt that some of the substance was swallowed: the man was seized after the meal with pain in the bowels and vomiting. He died in nineteen days. On inspection, no appearances indicative of poison were met with in the alimentary canal, and an opinion was given that the deceased had died from malignant fever. (Mémoires et Rapports, 330.)

SULPHATE OF ZINC. WHITE VITRIOL, OR WHITE COPPERAS.

This substance is ranked among irritant poisons, although it is certainly not very active as such. In doses of from a scruple to half a drachm, it is given as an emetic in most cases of poisoning; and as it frequently presents itself in the contents of the stomach in these cases, it is important that the medical jurist should be acquainted with its chemical properties. In order to show the slightly irritant effects of this substance, it may be stated that Dr. Babington of Guy's Hospital gave to a girl aged 17, thirty-six grains three times a-day for several weeks without any sickness or other untoward effect being produced. When the dose was raised to forty-two grains, which the girl continued to take for one week, she lost her appetite and felt much sickness (G. H. Rep. No. xii. p. 17.) This must be regarded as a somewhat unusual case. Orfila refers to two instances, wherein sulphate of zinc was taken in a pretty large dose; but both of the patients recovered: in fact, in general, the powerfully emetic properties of this substance interfere with its action as an irritant; since it is speedily expelled from the stomach by vomiting. Nevertheless, in four or five instances reported by continental writers, this poison has destroyed life; but there is, I believe, no instance recorded of its having operated fatally in England. It possesses a strong metallic taste, which is not easily concealed by any kind of food.

SYMPTOMS AND APPEARANCES.

The symptoms produced by an over-dose, are pain in the abdomen and violent vomiting, coming on almost immediately, and diarrhœa.

After death, the stomach has been found inflamed. The sulphate appears to act as a pure irritant; it has no corrosive properties.

Criminal cases of poisoning by zinc are very rare: hence the following which was recently tried in France is of some interest. An old man, aged 84, died somewhat suddenly, having suffered from severe pain and great heat in the chest and abdomen, with violent vomiting and purging. No medical man was called to see him. On inspection, the stomach and bowels were found highly inflamed, varying in colour from a pale-red to a deep red-brown. Sulphate of zinc was found in the contents of the viscera, and the metal was detected in the tissues, thus proving that it must have been administered during life. The inspectors assigned this as the cause of death. The body of a woman who had died two months previously, was then disinterred, and sulphate of zinc was found in the viscera. From circumstances which transpired, death was referred to the action of this irritant (Journal de Chimie Médicale, 1845. p. 529.) The parties who were charged with this double murder, were tried and acquitted for want of evidence. M. Chevallier mentions a case of compound poisoning in which a mixture of sulphate of zinc and arsenic, was administered and caused death.

TREATMENT.—Warm water, with milk, should be freely exhibited: it has been recommended to give albumen as an antidote, but it requires a very large quantity of this substance to precipitate the oxide of zinc: some have advised that albumen mixed with carbonate of magnesia should be given. All infusions containing tannin may be usefully exhibited, such as tea, oak-bark, or Peruvian bark, or these substances may be given in powder. A strong decoction of tea will equally answer. If the poison should have entered into the intestinal canal, a fact indicated by severe pain in the lower part of the abdomen, emollient enemata may be administered.

CHEMICAL ANALYSIS.—The pure sulphate is seen in white prismatic crystals, closely resembling in appearance, sulphate of magnesia and oxalic acid; from oxalic acid it is distinguished, by being fixed when heated on platina foil,—from the sulphate of magnesia, by tests applied to its solution. It is readily dissolved by water; this fluid taking up about one-third of its weight at common temperatures. Analysis of the *solution*.—The solution in water has a slightly acid reaction. The following tests may be used for the detection of oxide of zinc: 1. *Ammonia* gives a white precipitate, soluble in an excess of the alkali. 2, *Sesquicarbonate of ammonia*, a white precipitate, also soluble in a large excess of the test. 3. *Ferrocyanide of potassium*, a white precipitate. 4, *Sulphuretted hydrogen* and hydrosulphuret of ammonia, a white milky precipitate, provided the solution be *pure* and neutral, or nearly so. If the solution be very acid, sulphuretted hydrogen produces no effect whatever. These last-mentioned tests also throw down sulphuret of zinc from the precipitates dissolved by ammonia and its sesquicarbonate. It must be remembered that a solution of sesquicarbonate of ammonia, if kept in a flint glass, often contains lead, and is thus rendered brown by the sulphuretted hydrogen gas. 5. Sulphuric acid in the solution, is detected by the usual test, nitrate of barytes.

Delicacy of the tests.—In a single drop of water, either of the three last-mentioned tests will produce a slight effect with the 1-440th grain of sulphate of zinc. Dilution materially affects their action. They are nearly equally delicate, but the action of neither was apparent with less than *one-quarter of a grain* of sulphate, when this was diffused through twelve ounces of water: and as all the precipitates are white, the effects in this diluted state are by no means characteristic or satisfactory.

Objections.—Ammonia gives a white precipitate with numerous solutions—as, with those of magnesia, the salts of lead, cadmium and the persalts of mercury; but it redissolves only the precipitate from zinc. Sesquicarbonate of ammonia precipitates many salts, white; but it is only the precipitate from zinc, which it has the power of redissolving. This test, by giving no precipitate with sulphate of magnesia, clearly distinguishes that salt from sulphate of zinc. Ferrocyanide of potassium is a delicate test, since it will show the presence of the sulphate of zinc when forming only the 40,000th part of a solution: but it precipitates numerous other metallic salts white; and is therefore only a corroborative test. The action of sulphuretted hydrogen is perfectly characteristic of zinc; since this is the only metal, the salts of which are thrown down white by it. In order to precipitate it effectually, the oxide of zinc should be first precipitated and redissolved by ammonia, and the gas then passed into the alkaline solution. The common *White vitriol* of commerce is in rough reddish-white irregular semicrystalline masses. When dissolved in water, the action of the tests is somewhat different, because this substance usually contains iron. Thus, oxide of iron is left on redissolving the precipitate given by ammonia and its sesquicarbonate; the precipitate from ferrocyanide of potassium is blue or blueish-white, instead of white; and the sulphuret thrown down by the fourth test, is of a dark-brown colour. Among the common salts which might be mistaken for zinc in solution, is *alum;* for this last is precipitated by all the tests above mentioned, except sulphuretted hydrogen gas and ferrocyanide of potassium. It strongly resembles a metallic solution, in being precipitated by hydrosulphuret of ammonia, owing to the alkali separating alumina; but the non-precipitation by ferrocyanide of potassium, and the insolubility of the precipitated alumina in the ammoniacal tests, would easily distinguish a solution of alum.

In organic mixtures.—If the sulphate of zinc be dissolved, we may pass into the solution, a current of sulphuretted hydrogen gas; the presence of zinc is immediately indicated by a milky-white froth—the sulphuret may be collected, and decomposed by boiling it with muriatic acid. The white sulphuret of zinc is apt to conceal faint traces of arsenic, when the sulphate has been given as an emetic in cases of arsenical poisoning. Reinsch's test may then be used to detect the presence of that poison: the sulphuret of arsenic is soluble in ammonia, and may thus be separated from that of zinc; but a few drops of muriatic acid will answer better for this separation: this acid converts the sulphuret of zinc to a soluble chloride, but scarcely affects the sulphuret of arsenic. Hence, if the compound liquid be strongly acidulated with muriatic acid, the sulphuret of arsenic only is precipitated by a current of sulphuretted hydrogen gas. If too much muriatic acid be used, the necessary neutralization by ammonia produces so much muriate of ammonia as to prevent the action of the tests,—the zinc-precipitates being soluble in this salt. The analyst must remember that zinc sometimes contains traces of cadmium; and this has been known to give rise to a wrong suspicion of the presence of arsenic in zinc and its compounds.

Zinc in the tissues.—If the salt of zinc be decomposed, and we have to search for it in the mucous membrane of the stomach, this may be cut up and boiled in diluted nitric acid; if necessary, the nitrate may be then neutralized by ammonia, and thrown down as sulphuret by a current of sulphuretted hydrogen gas. The viscera may be also incinerated with flux, and the zinc procured in the metallic state, or dissolved out of the residue by muriatic acid.

Quantitative analysis.—The zinc should be converted to oxide, every one hundred parts of which are equal to three hundred and fifty-seven parts of crystallized sulphate.

CARBONATE OF ZINC, (CALAMINE.)

This compound does not appear to have any poisonous action; and it would probably require to be given in large quantity to produce any effect. Carbonate of zinc is the white substance which is formed on the metal when long exposed to air and moisture. Its effects may become a subject of investigation as a matter of medical police; since zinc is now much used for roofing, and also in the manufacture of water-pipes and cisterns. (Ann. d'Hyg. 1837, 281, ii. 352.)

Chemical analysis.—The pure carbonate is a white-powder, insoluble in water: it possesses a faint alkaline reaction. It becomes yellow when heated, and undergoes no change in hydrosulphuret of ammonia. It is dissolved with effervescence by diluted sulphuric acid; and in this state, the tests for zinc may be readily applied to the solution. Calamine is an impure carbonate destitute of poisonous action. It consists of carbonate of zinc, silicate of zinc and peroxide of iron, which gives it a red colour. Some specimens have been found to consist in great part of sulphate of barytes. One specimen, according to Dr. Thomson contained eighty-eight per cent. of sulphate of barytes,—the rest consisted of oxide of iron, alumina and chalk. (See Lancet, June 8, 1844, p. 345.)

There was not a trace of zinc or lead in it. The *active* principle of the calamine ointment of the old pharmacopœias must therefore have been the *lard!*

Action of water and other liquids on zinc.—Zinc is largely employed for the purposes of roofing—for gutters, cisterns, and pipes, through which water is circulated. I have found by experiment that pure water, under access of air, has a very rapid action on zinc. On leaving a polished plate of the metal in *distilled water* for forty-three days, it was rendered turbid by a flocculent deposit consisting of carbonate of zinc. About three ounces of the water yielded by filtration two grains of carbonate of zinc. A similar experiment performed with *river water* gave a much smaller quantity of carbonate. The zinc, when removed, was covered with a white gelatinous film, and had lost its polish. The presence of alkaline chlorides in water leads to a slow chemical action in which soluble chloride of zinc is formed. Any acid present in water accelerates the chemical action. (See Ann. d'Hyg. 1837, i. 281; ii. 352.)

In repeating these experiments with sheet-iron coated with zinc (galvanised iron) similar results were obtained. Water collected in zinc-vessels or transmitted through zinc-pipes, is therefore not adapted to use, unless it has undergone filtration; but even in this case, a minute quantity of hydrated oxide or carbonate of zinc may still be retained by it in a state of solution.

When an *acid* liquid has been placed in a zinc-vessel, there is a strong chemical action, and the liquid becomes invariably impregnated with a salt of zinc. A cider merchant kept for three months a quantity of cider in vessels made of zinc. It was observed that the liquid had then acquired an acrid and styptic taste. On analysis it was found to contain a large quantity of acetate of zinc. It had, therefore, become decidedly poisonous. (Gaz. Méd. 14 Nov, 1846, p. 905.)

CHLORIDE OF ZINC.

This is a very soluble deliquescent salt; the chlorine is detected by nitrate of silver.

ACETATE OF ZINC.

This is a white crystalline salt of zinc, but very little used. It may be mistaken for sulphate of zinc, especially as it often contains some of this salt. It is known by boiling it in diluted sulphuric acid, when the acetic acid is expelled and identified by its odour.

LACTATE OF ZINC.

Zinc has been lately used in making utensils for holding milk during the separation of cream. It is probable that some of this salt is here formed, as well as a combination of oxide of zinc with casein. I have been informed that milk and cream which were allowed to stand in such vessels, have given rise to nausea and vomiting. This practice would not be allowed under a proper system of medical police.

OXIDE OF ZINC.

This substance is not very active, but when taken for a long continuance, it may produce serious symptoms. The following singular case of slow poisoning by the oxide is reported in the Brit. and For. Med. Rev. No. xi. p. 221. A man, aged forty-five, wishing to treat himself for epilepsy, took twenty grains of oxide of zinc daily, until he had taken the enormous quantity of three thousand two hundred and forty-six grains! He was then seen by a physician;

he was considerably emaciated,—his bowels constipated, the extremities cold, the legs œdematous, the abdomen tumid, the skin dry—and the pulse slow and scarcely perceptible. Under proper treatment he recovered. This substance is known by its becoming of a yellow colour when heated, and by its ready solubility in diluted acids, with the action of the tests for zinc upon the solution.

CHAPTER XXX.

ACTION OF THE CHLORIDES OF TIN—DYER'S SPIRIT—ANALYSIS—POISONING BY LUNAR CAUSTIC—TREATMENT—ANALYSIS—TERCHLORIDE OF GOLD—ANALYSIS—SULPHATE OF IRON, GREEN VITRIOL—MURIATE OF IRON—ANALYSIS—SUBNITRATE OF BISMUTH—ANALYSIS—POISONING BY BICHROMATE OF POTASH—ITS LOCAL ACTION—ANALYSIS—THE SALTS OF PLATINA, PALLADIUM, IRIDIUM, RHODIUM, OSMIUM, NICKEL, MANGANESE, CERIUM, URANIUM.

PREPARATIONS OF TIN.

Tin in the metallic state, as in the form of *Tin filings*, is not poisonous: but it is proper to state that, according to M. Chevallier, English tin contains 1-775th part of arsenic. The only preparations of this metal, which require to be noticed as poisons, are the Chlorides, or Muriates, a mixture of which is extensively used in the arts, under the name of Dyer's Spirit. The salts may exist in the form of whitish-yellow crystals; but more commonly they are met with in a strongly acid solution in water. They are irritant poisons; but so seldom used as such, that only one death occurred from them in England and Wales during a period of two years. They are decomposed by magnesia and many organic principles; and this alkali, with milk or albumen, should be freely used in treating a case of poisoning by them.

Chemical analysis. Protochloride.—In the solid state it forms a milky solution with water, which disappears on the addition of muriatic acid. The acid solution in water is characterized by the following properties: 1. *Chloride of gold* gives a deep purple-brown precipitate, almost black. 2. *Bichloride of mercury*, in small quantity, gives a white, passing to a grey, precipitate of metallic mercury. 3. *Sulphuretted hydrogen* gas or hydrosulphuret of ammonia, gives a deep chocolate-brown precipitate, even in diluted solutions. 4. *Nitrate of silver* gives a white precipitate insoluble in nitric acid, thus proving the presence of muriatic acid or chlorine.

Bichloride or permuriate.—This is commonly met with as a highly acid liquid; it is not precipitated by chloride of gold or bichloride of mercury. It is known from other metallic poisons, except arsenic and cadmium, by giving a yellowish precipitate with *sulphuretted hydrogen* gas. It is known from an arsenical liquid, among other properties, by this yellow precipitate being insoluble in a small quantity of ammonia, and from cadium by the precipitate not being dissolved by muriatic acid. When heated with black flux, it yields no metallic sublimate. The solution is also precipitated of a yellow-brown colour, by the *hydrosulphuret of ammonia*. Nitrate of silver will detect the acid. If we have to search for these poisons in the stomach, the better way will be to boil the solids in strong muriatic acid. This dissolves out in great part the oxide of tin. If this should fail, the viscera may be dried and calcined with three or four parts of black flux, when metallic tin may be obtained by washing the residue.

PREPARATIONS OF SILVER.

Nitrate of silver. Lunar caustic. Lapis infernalis.—This substance, which is commonly met with in small sticks of a white or dark grey colour, is readily soluble in distilled water; in common water it forms a milky solution. It acts as a powerful corrosive, destroying all the organic tissues with which it comes in contact. There are at least two cases on record, in which it has proved fatal in the human subject:—one of these occurred in 1837-8; but the particulars are unknown. The symptoms come on immediately, and the whitish flaky matter vomited, is rendered dark on exposure to light. Coloured spots on the skin will also indicate the nature of the poison. The treatment consists in the administration of magnesia and common salts with emetics.

Chemical analysis.—The *solution* in water is commonly acid. 1. A slip of *copper* introduced into a small quantity, precipitates metallic silver. 2. *Muriatic acid* throws down a white clotted precipitate of chloride of silver, insoluble in nitric acid, but soluble in ammonia and the alkaline hyposulphites. 3. *Arsenite of ammonia* gives a yellow precipitate. 4. *Arsenic acid* a brick-red precipitate. 5. *Sulphuretted hydrogen*, and the hydrosulphuret of ammonia, a black precipitate. 6. The nitric acid is discovered by adding carbonate of potash, when the filtered liquid will be found to contain nitre. This poison is absorbed,—a fact made evident when it is given for a long time in small doses by the discoloration of the skin. It has also been found in the tissues of animals in acute cases of poisoning. Although absorbed, absorption is not necessary to its action as a poison.

PREPARATIONS OF GOLD.

Terchloride.—This is the only preparation of gold which requires notice. It is a powerful irritant poison, acting locally like the nitrate of silver. Nothing is known of its effects on the human subject, but in administering it to animals, Orfila has found extensive inflammation and even ulceration of the mucous membrane of the stomach. (Toxicologie, ii. 30.) The metal is absorbed and carried into the tissues, but its poisonous action is wholly independent of absorption.

Treatment.—Magnesia and albumen.

Analysis.—The solution is of a rich yellow colour, acid, and it stains organic matter purple. 1. In a very diluted state *Chloride of tin* produces a deep purple precipitate, the colour varying according to the degree of concentration. 2. *Oxalic acid* or *sulphurous acid* in excess speedily precipitates metallic gold in reddish-coloured crystals. 3. A solution of phosphorus in alcohol, also precipitates the metal of a dark colour, in a finely-divided state. 4. *Sulphuretted hydrogen gas* produces a deep chocolate-coloured precipitate, almost black, of sulphuret of gold. This solution differs from those of most other metals, in not giving a precipitate with the ferrocyanide of potassium. (See table, ante, p. 131.)

PREPARATIONS OF IRON.

It seems that, although the oxide and carbonate of iron may be given in very large quantity, without any serious effects resulting, yet some of the saline preparations of this metal act as irritants.

Sulphate of iron. Copperas. Green vitriol.—This compound has been several times administered with malicious intention. (See ante, p. 17.) One death from this substance took place in 1837-8. It cannot, however, be a very active preparation; for a girl who swallowed an ounce of it, recovered, although she suffered for some hours from violent pain, vomiting and purging. (Christison on Poisons, 506.) A remarkable case is related by the same writer, in which it was highly probable that this salt occasioned the death of a girl, four years of age. The deceased had been previously in good health, but she was attacked with vomiting and purging after her breakfast, and died the same afternoon. Four months afterwards, the body was disinterred and examined. The œsophagus, stomach, and duodenum were soft, gelatinous, and of an intensely black colour through their whole substance. This was especially observed in the stomach. The whole of the alimentary canal, from the mouth to the anus, was lined throughout with a jet-black mucus. A suspicion having arisen that sulphate of copper had been given, the black substance was examined for this metal, but none was found. On further analysis it was ascertained to consist of iron in the state of sulphuret, and the contents were found to give evidence of the presence of a much larger quantity of sulphuric acid than is usually present. Dr. Christison very justly inferred, that a soluble preparation of iron had been given before death, since, although iron is contained in all kinds of organic matter, it is only in minute traces. It is proper to mention here that for a long period after interment, the whole of the stomach and alimentary canal may be found intensely black on the mucous surface, irrespective of the presence of any metallic poison.

Green vitriol or copperas is sometimes given as an abortive. A suspicious case is reported, in which a woman far advanced in pregnancy, but enjoying good health, was suddenly seized about midnight with vomiting and purging, and died in fourteen hours. The body was disinterred, and iron found in large quantity in the viscera. The symptoms are not always of this violent kind. In a case which occurred to M. Chevallier, a husband gave a large dose of sulphate of iron to his wife. There was neither colic nor vomiting. The woman lost her appetite, and she had a clayey complexion, but she ultimately recovered.

External application—A case which seems to show that this substance may really act through the skin, has been lately reported by Mr. Moore, of York. A healthy boy, aged fourteen after having been employed in picking crystals from the vat in which sulphate of iron was set to crystallize, was attacked with headach and sickness. He vomited several times, felt pains in the calves of his legs, and colicky pains in the abdomen; at the same time his limbs became contracted. The boy had previously complained that the liquor of the crystals, into which he was constantly dipping his hands, had cracked his fingers. In the course of a week or ten days, these symptoms disappeared under treatment. (Med. Gaz. XXX. 351.) No other cause could be assigned for this singular attack, than the frequent contact of the hands with the saturated solution of the green sulphate of iron. I have known a similar dryness and cracking of the skin of the hands produced by contact with a strong solution of sulphate of copper; and the dyers of Glasgow are said to have been attacked at one time with troublesome sores on the hands, from the frequent immersion of them in a solution of bichromate of potash. (See p. 406.)

CHEMICAL ANALYSIS.—This substance is generally met with in crystals of a sea-green colour. It is readily soluble in water. 1. *Ferrocyanide of potassium* added to the solution, gives a greenish blue precipitate, becoming of a deep blue by exposure to the air. 2. *Sulphocyanide of potassium* gives a reddish precipitate, which, after a short exposure to the air, becomes of a deep blood-red colour. 3. *Hydrosulphuret of ammonia* gives a black precipitate. 4. Nitrate of barytes will show the presence of sulphuric acid. With the PERSULPHATE of iron, these tests produce immediately the changes of colour indicated.

MURIATE OF IRON. TINCTURE OF SESQUICHLORIDE OF IRON,—This is an acid solution of peroxide of iron with alcohol, of a red colour, much used in medicine. Dr. Christison relates an instance, where a man, by mistake, swallowed an ounce and a half of this liquid: symptoms were somewhat like those produced by muriatic acid. He at first rallied, but died in about five weeks. The stomach was found partially inflamed, and thickened towards the pylorus. A case was reported to the Westminster Medical Society, in November 1842, where a girl, aged fifteen, five months advanced in pregnancy, swallowed an ounce of the tincture of muriate of iron in four doses in one day, for the purpose of inducing abortion. Great irritation of the whole urinary system followed; but this was speedily removed, and she recovered. Another case of recovery from a large dose of this preparation has been recently reported by Mr. Amyot. A healthy married female swallowed by mistake for an aperient draught, *one ounce and a half* of the tincture of muriate of iron. She immediately ejected a portion, and violent retching continued for some time. There was great swelling of the glottis, cough, with difficulty of swallowing. These symptoms were followed by heat and dryness of the throat, with a pricking sensation along the course of the œsophagus and stomach; and in the afternoon a quantity of dark grumous blood was vomited. The motions were black, owing doubtless to the action of sulphur upon the metal. In about a month the patient was perfectly restored to health. (Provincial Journal, April 7 and 21, 1847, 180.)

TREATMENT.—In poisoning by either salt of iron, magnesia or the alkaline carbonates should be freely given.

CHEMICAL ANALYSIS.—The muriatic acid may be detected by nitrate of silver and nitric acid, while the peroxide of iron is immediately indicated by a precipitate of Prussian blue on adding a solution of *Ferrocyanide of potassium*. The *Sulphocyanide of potassium* gives a deep blood-red colour; and the hydrosulphuret of ammonia a black precipitate. The ferrocyanide of potassium gives a green tint when the iron is in small quantity, when the liquid is much diluted, and the solution is very acid.

PREPARATIONS OF BISMUTH.

SUBNITRATE OF BISMUTH. PEARL-WHITE. MAGISTERY OF BISMUTH.—This substance, in a dose of *two drachms*, caused the death of an adult in nine days. There was burning pain in the throat, with vomiting and diarrhœa,—coldness of the surface, and spasms of the extremities,—also a strong metallic taste in the mouth. On inspection, the fauces, larynx, and œsophagus were found inflamed; and there was inflammatory redness in the stomach and throughout the intestinal canal. (Sobernheim, 335.) In a case mentioned by Dr. Traill, a man took by mistake *six drachms* of the subnitrate, in divided doses, in three days. He suffered from vomiting, and pain in the abdomen and throat, but finally recovered. (Outlines, 115.) These cases are sufficient to prove that a substance very slightly soluble in water, may exert a powerfully poisonous action on the human system.

CHEMICAL ANALYSIS.—This substance is commonly seen under the form of a white powder, or occasionally in semitransparent pearly crystals. 1. The powder is blackened by *Hydrosulphuret of ammonia*, in which respect it resembles a salt of lead. 2. It is turned of a yellow colour, passing to a deep chocolate-brown, with *Iodide of potassium*. It is scarcely soluble in water, but readily dissolves in nitric acid; and when this solution is added to a very large quantity of water, the subnitrate, if the liquid be not too acid, is again precipitated. In this respect it resembles only chloride of antimony; but it is known from this compound by the action of sulphuretted hydrogen, hydrosulphuret of ammonia. (See p. 397.)

PREPARATIONS OF CHROME.

The only compound of chrome which requires any notice as a poison is the Bichromate of Potash.

This salt is extensively used in the art of dyeing; and one death is reported to have occurred from it in 1837–8, but the particulars are unknown. There is no doubt that it is an irritant as well as a corrosive poison, affecting also powerfully the nervous system. In animals it has produced in small doses vomiting, diarrhœa, paralysis, and death in a few hours. It appears that, like some other metallic salts, it has a local action when in a state of concentrated solution; and thus it has been observed, in some cases, to produce extensive sores on the hands of dyers, owing to frequent contact with the liquid. According to Dr. Baer this is only likely to happen when the skin is abraded; but the skin may be slowly destroyed by the salt. (Beck's Med. Jur., 823.)

Well-observed instances of poisoning by this compound, which is now extensively used in the arts, are rare; and, therefore, the details of the following case, communicated to the Medical Gazette (xxxiii. 734) by Mr. Wilson of Leeds, are of great practical interest. A man, aged sixty-four, was found dead in his bed, twelve hours after he had gone to rest. He had been heard to snore loudly during the night, but this had occasioned no alarm to his relatives. When discovered, he was lying on his left side, his lower extremities being a little drawn up to his body: his countenance was pale, placid, and composed; eyes and mouth closed; pupils dilated; no discharge from any of the outlets of the body; no marks of vomiting or diarrhœa, nor any stain upon his hands or person, or upon the bed-linen or furniture. The surface was moderately warm. Some dye-stuff, in the form of a black powder, was found in his pocket. On inspection, the brain and its membranes were healthy and natural; there was neither congestion nor effusion in any part. The thoracic viscera were equally healthy, as well as those of the abdomen, with the exception of the liver, which contained several hydatids. A pint of turbid inky-looking fluid was found in the stomach. The mucous membrane was red and very vascular, particularly at the union of the cardiac extremity with the œsophagus: this was ascribed to the known intemperate habits of the deceased. In the absence of any obvious cause for death, poison was suspected; and on analysing the contents of the stomach, they were found to contain bichromate of potash;—the dye-powder taken from the man's pocket, consisted of this salt mixed with cream of tartar and sand. It is remarkable that in this case there was neither vomiting nor purging. The salt does not appear to have operated so much by its irritant properties, as by its indirect effects on the nervous system. This, however, is by no means an unusual occurrence, even with irritants far more powerful than the bichromate of potash. The following case occurred to Dr. Baer of Baltimore. A man in drawing off a solution of the bichromate by a syphon, accidentally received a small quantity into his mouth. In a few minutes he perceived great heat in the throat and stomach, and this was followed by violent vomiting of blood and mucus. The vomiting continued incessantly until his death, which took place in *five hours*. On dissection, the mucous membrane of the stomach, duodenum, and about one-fifth of the jejunum, was destroyed in patches. (Beck's Med. Jur., 823.) In this instance the salt acted as an irritant. The two cases here given, show that the bichromate of potash is a very active poison; and, as it is largely employed in the arts, it deserves the attention of medical jurists.

Dr. Berndt observed that thirty grains of the bichromate, introduced into a wound on the back of a dog, produced vomiting, paralysis of the hinder extremities, and death in eleven hours. This gentleman found that the CHROMATE was as active a poison as the BICHROMATE, but that the PROTOXIDE (green oxide) of Chromium was inert. (See Brit. and For. Med. Rev. April 1839, xiv. 506.)

TREATMENT.—Besides emetics, carbonate of magnesia or chalk, mixed up in a cream with water, should be given.

CHEMICAL ANALYSIS.—This is an acid salt, easily known from all the other metallic poisons, by its being in crystals of a deep orange-red colour. It is readily soluble in water, and the solution has the rich orange-colour of the salt. It has an acid reaction. It may be identified by the following tests:— 1. The solution is precipitated of a rich red colour, by *Nitrate of silver*. 2. Of a bright yellow, by the *Acetate of lead*. 3. Of a dingy green, by a current of *Sulphuretted hydrogen gas*. Potash may be discovered in it by the action of chloride of platina.

[Several fatal cases of accidental poisoning with this salt have occurred in the United States, especially at Baltimore, where it is manufactured on a large scale. Dr. Ducatel has fully treated on this subject. (Man. Toxicol. 144.) He advises the administration of carbonate of potash or soda to neutralize the excess of chromic acid, as the best primary treatment, followed by emetics.—G.]

SALTS OF PLATINA, PALLADIUM, AND OTHER METALS.

The salts of PLATINA, PALLADIUM, IRIDIUM, RHODIUM, OSMIUM, COBALT, NICKEL, MANGANESE, CERIUM, and URANIUM, also possess an irritant action, partly depending on the acids with which they are combined. According to the experiments of Gmelin on animals, the OXIDE OF OSMIUM appears to be the most active poison among them. They are products of art not met with in common life; and, so far as I can ascertain, they have never been taken as poison by man. It is unnecessary, therefore, to occupy space by detailing the chemical processes whereby they may be identified; these will be found fully described in all works on chemistry. This concludes the history of the MINERAL IRRITANT POISONS.

VEGETABLE IRRITANTS.

CHAPTER XXXI.

DIVISION OF VEGETABLE POISONS—MODE OF ACTION OF VEGETABLE IRRITANTS. ALOES. ANEMONE. ARUM MACULATUM. BRYONY. CAYENNE PEPPER. CELANDINE. COLOCYNTH. DAFFODIL. ELATERIUM. ELDER. EUPHORBIUM. HYSSOP. JATROPHA. CURCAS. JALAP. MANCHINEEL. MEZEREON. MUSTARD. RANUNCULUS. SAVIN. SCAMMONY. SORREL. STAVESACRE.

The poisonous substances of an irritant nature, which belong to the vegetable kingdom, are very numerous as a class; but it will here be necessary to notice only those which have either caused death, or given rise to accidental poisoning.

The true vegetable irritants, soon after they are swallowed, produce severe pain in the abdomen, accompanied by vomiting and diarrhœa. There are rarely any cerebral symptoms, and no convulsions: the occurrence of the former would place them in the class of NARCOTICS and of the latter in that of the NARCOTICO-IRRITANTS.

It must be admitted, however, that the operation of many of them is by no means clearly defined. Stupor, delirium, and convulsions are occasionally observed: hence the distinction between some vegetables, here placed among irritants, and those which are assigned to the narcotico-irritant class, is purely arbitrary. Further experience may hereafter lead to a better knowledge of their *modus operandi*, and to an improved classification. One circumstance is worthy of remark. The effects of narcotico-irritant poisons can commonly be traced to the presence of a poisonous alkaloid in the vegetable. Among the irritants, the effects appear to be principally due to the presence of an acrid oil or resin. There is only one, the *Delphinium Staphysagria*, in which an alkaloidal principle, *Delphinia*, has been found to exist.

Some of the vegetable irritants act especially on the bowels, and, in mild doses, are safely used as purgatives. In large doses they produce hypercatharsis, and in old and young subjects are apt to cause death by exhaustion. There are, however, but few instances recorded of their fatal action on the human body; and the little that is known concerning their operation as poisons, is chiefly derived from the experiments performed by Orfila on animals. The changes found after death are confined to irritation and inflammation of the alimentary canal. These substances (if we except SAVIN) are rarely resorted to by the suicide or murderer,—for large doses are required, and their fatal operation even in these cases is rendered uncertain by the circumstance that they excite vomiting, and are then commonly expelled from the stomach.

TREATMENT.—In cases of poisoning by the vegetable irritants, emetics should be freely employed, and when the poisonous vegetable is expelled, antiphlogistic measures may be used. If the seat of pain should indicate that the poison has reached the bowels, purgatives or cathartic enemata may be administered. The strength of the individual should be supported.

A selection of the vegetable irritants will be here given in an alphabetical order, as this appears better adapted for practical purposes than any botanical arrangement.

The following is a list of some of those vegetable substances which are considered to act as irritant poisons. Aloes, Anemone, Arum, Bryony, Capsicum, Castor Seeds, Celandine, Colocynth, Creasote, Croton Seeds and Oil, Daffodil, Elaterium, Elder, Euphorbium, Gamboge, Hyssop, Jatropha, Jalap, Manchineel, Mezereon, Mustard, Ranunculus, Savin, Scammony, Stavesacre, Oil of Tar, Oil of Turpentine, Decayed Vegetable Matter.*

ALOES.

This well-known inspissated juice of several varieties of plants, acts as a purgative in doses varying from five to twenty grains. When given in larger doses or frequently repeated it excites violent purging. It requires often many hours for its operation: it is less irritating than jalap or scammony, and it appears to act especially on the large intestines.

Aloes, mixed with gamboge and colocynth, are said to be the basis of a certain quack-medicine, sold under the name of Morison's Pills. These have proved fatal in many instances from the exhaustion produced by excessive purging, owing to the large quantity of these pills, taken in frequently-repeated doses. Our knowledge of the symptoms and post-mortem appearances produced by these irritants, is, indeed, chiefly derived from the cases which have proved fatal under this pernicious treatment. In the seventeenth volume of the Medical Gazette, will be found four cases of this description. The most prominent symptom was excessive diarrhœa, with the discharge of large quantities of mucus and blood; the individual became emaciated, and slowly sank from exhaustion. In some instances, the symptoms are those of inflammation and ulceration of the bowels. In 1836, a man was convicted of having caused the death of a person by the administration of these pills; in this instance the death of the deceased was clearly due to the medicine,—and on inspection, the stomach was found inflamed and ulcerated; the mucous membrane of the small intestines was injected and softened, and there was the appearance of effused lymph upon it. An ingenious attempt was made in the defence to draw a statement from the medical witness, that the good effects of some medicines invariably increased in proportion to the quantities taken!—this antihomœpathic proposition was, however, very properly rejected. In all cases, it must be remembered, that these drastic purgatives may cause serious symptoms, or even death, when administered to young infants, or to persons debilitated by age or disease; nor is it necessary that the dose should be very large for fatal effects to follow. The medical question here may be, whether the medicine caused death directly, or whether it simply accelerated it. Hicrapicra appears to be a popular aloetic compound, and one death is recorded to have been produced by this in 1837-8. In another instance death was caused by an individual taking aloes in nitric acid, in which case the mineral acid was most probably the destructive agent. A singular case occurred in Germany a few years since, wherein a medico-legal question was raised respecting the poisonous properties of aloes. A woman, aged 43, not labouring under any apparent disease, swallowed *two drachms* of powdered aloes in coffee. Violent diarrhœa supervened, and she died the following morning, twelves hours after having taken the medicine. On inspection, the stomach was found partially, and the small intestines extensively, inflamed. There

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[* For descriptions and accounts of properties of these plants, see Griffith's Medical Botany.]

were no other particular appearances to account for death, and this was referred to the effect of the aloes.

This case appears to show that aloes possesses an irritant action. A large dose, given to a person debilitated by disease, might easily cause death, as the result of exhaustion from hypercatharsis.

Analysis.—Powdered aloes has a snuff-brown colour. When heated it gives off a thick vapour, having the peculiar odour of this substance: it melts and burns with a smoky flame, leaving an abundant carbonaceous ash. Strong nitric acid dissolves it, and acquires a rich red-brown colour. Sulphuric acid gives with it a yellow-brown colour:—a persalt of iron, a deep purple-black. It is soluble in water and alcohol; the solution is slightly acid, and has an intensely bitter persistent taste.

ANEMONE.

This is a genus of plants comprising several species, all possessed of irritating properties in the moist state, but which they appear to lose in great part when dried or exposed to heat, owing to the presence of a volatile principle, *Anemonine*. These plants have a strong acrid burning taste, which is stronger in the root than in the leaves. The Anemone Pulsatilla (Wind-Flower,) and Anemone Pratensis, are the two principal varieties. Small doses of the extract of the latter produced, according to Stork, an increased flow of urine, pain in the abdomen, and diarrhœa. The different parts of these vegetables have a local irritant action. All that is known concerning their operation on the human subject, is comprised in the following cases. Haller and Böckler remarked that they caused vesication of the skin, and that the distilled water produced nausea and vomiting. Orfila relates that an apothecary suffered from irritation of the eyes, colic, and vomiting, after having bruised some anemone pulsatilla. (Toxicologie, ii. 133.) Bulliard reports the case of a man who applied the bruised leaves of the plant to the calf of his leg. There was great pain for ten or twelve hours, and the local irritation was so severe that inflammation and gangrene followed. (Orfila, ib.; also Wibmer Die Wirkung der Arzneimittel, i. 178.) No instance is recorded of the plant having destroyed human life, but experiments on animals show that it will act fatally like other irritants; and that it causes most violent inflammation in all parts of the alimentary canal. In some instances symptoms indicative of an affection of the nervous system appeared.

Analysis.—The nature of this poison can only be determined by the botanical characters of the plant.

ARUM MACULATUM.

This is a well-known hedge-plant, the juice of which possesses irritating properties, which appear to depend on a volatile principle, as they are lost on desiccation or distillation with water. The plant has a local irritant action; but a sharp hot taste is only perceived after some time. The leaves appear to be the most acrid parts of the plant. In an instance reported by Bulliard, three children ate of the leaves: violent convulsions supervened,—one child died in twelve and another in sixteen days; the third child, after suffering from diarrhœa, recovered. It was observed that the tongue became so swollen in these cases that there was great difficulty of deglutition. (Wibmer, Op. cit., i. 338.) Dr. Christison states that he has known acute burning pain of the mouth and throat, pain of the stomach, vomiting, colic, and some diarrhœa, occasioned by eating two leaves. (On Poisons, 602.) Orfila found that the fresh root killed dogs in from twenty-four to thirty-six hours:—the intestinal canal was found inflamed.

ANALYSIS.—This poisonous plant can only be recognised by its botanical characters: it is very commonly found in hedge-rows during spring, and is vulgarly known in some counties under the name of Cuckow-pint or Wake-robin.

[A common species in the United States, the A. TRIPHYLLUM, is possessed of the same properties. See Griffith's Med. Bot. 616.—G.]

BRYONY.

The roots of black and white Bryony contain a bitter principle, *Bryonine*, which is soluble in water, and to which they appear to owe their violent irritating properties. The powdered root when swallowed produces severe pain, vomiting and purging, and after death the stomach and intestines are found highly inflamed. Two cases of the poisonous action of black bryony on the human subject are recorded. In one a female recently delivered, had been ordered by her medical attendant to take an ounce of the black bryony root in a pint of water, and to have an injection of a concentrated decoction of the root. She died in four hours, and on examining the motions, it was found that the lining membrane of the rectum had been passed with them. Inspection of the body was not allowed. (Orfila, ii. 82.) A second case is quoted by Dr. Christison, in which a man took two glasses of an infusion of the root to cure ague. He was seized with violent tormina and diarrhœa, which nothing could arrest, and which soon terminated fatally. (Op. cit. 594.) Dr. Pereira saw a case of poisoning by black bryony in which the symptoms were those of cholera. The woman recovered. (Mat. Med. ii. 1509.)

The black bryony (BRYONIA NIGRA) is seen in large irregular pieces: these are sometimes of considerable size, and so distorted as to present some appearance to the human figure. The herbalists sell it under the name of Mandrake root: but the true name Mandrake or *Mandragora officinalis* is a narcotico-irritant poison. The black bryony root is sometimes employed by quacks as an internal medicine, and is therefore liable to occasion accidents. It is frequently applied externally to promote the absorption of extravasated blood.

The black bryony grows abundantly in hedges, with a long creeping stem: it is often called the wild vine,—it produces in the autumn greenish berries, which slowly acquire a dull red colour.

CAPSICUM.

CAPSICUM, under the form of CAYENNE PEPPER, is reported to have destroyed life in one case in 1837-8, but the particulars are not stated. This substance owes its hot taste and irritant properties to an acrid soft resin, *Capsicin* (a compound of resin and essential oil.) When the powder or the oil is taken in large doses, it produces severe pain, followed by inflammation of the œsophagus and stomach. In powder it is well known to have a peculiar smell and a very hot taste: this is owing to the volatility of the capsicin. Its local action is such that it will produce vesication of the skin. With the exception of the case above mentioned, I have never heard of any accident arising from its use. It may be stated that the medicinal dose of the powder is from five to ten grains,—of the tincture from ten minims to one drachm. The powder would be known by its insolubility, its red colour, its odour, and hot burning taste.

CELANDINE.

All that we know of this vegetable irritant (Chelidonium Majus) is derived from the experiments of Orfila on dogs. The aqueous extract produced severe symptoms of irritation, followed by death,—the poisonous principle appears to become after a time absorbed, and to act on the nervous system. After death the mucous membrane of the stomach was found intensely inflamed. (Op. cit. ii. 118.)

COLOCYNTH.

This substance, known as Bitter Apple, is used in the form of powder, extract, or decoction. It is properly ranked among vegetable irritant poisons, as it has caused death in several instances. In whatever form it is taken, it produces, in large doses, severe pain in the stomach and bowels, with the most violent purging,—the stools being mixed with mucus and blood. After death the mucous membrane of the stomach and intestines has been found highly inflamed. From the cases collected by Wibmer, it would appear that *one drachm* of colocynth administered in a clyster caused death. (Op. cit. i. 227.) Dr. Christison quotes the case of a female who died after incessant vomiting and purging from a dose equal to a teaspoonful and a half. (On Poisons, 595.) The extract of three apples caused bloody diarrhœa and death, and in another instance bloody stools, with spasms in the limbs, were produced by a single apple: the individual with difficulty recovered. In one fatal case mentioned by Orfila, the patient swallowed two glasses of a decoction of colocynth,—colic, burning pain in the bowels, diarrhœa, and intense thirst followed: the symptoms somewhat resembled those caused by arsenic. The man sank under the effects in less than four days, and on inspecting the body, the intestines were found glued together, inflamed, and covered with black spots: the mucous membrane of the stomach was detached and ulcerated. The liver, kidneys, and bladder were also inflamed. (Toxicol. ii. 93.) The substance which is extensively sold to the public in shops under the name of *Pill. Cochiæ*, is according to Dr. Pereira, a spurious extract of colocynth mixed with gamboge. (ii. 1676.)

The medicinal dose of the powder is from two to ten grains; of the extract from five to twenty grains.

Analysis.—The preparations of colocynth have an intensely bitter taste. This is owing to the presence of a bitter principle (*Colocynthin*) which forms a large proportion of the watery extract.

The *powder* is of a light brown colour—changed to a dark brown (artificial tannin) by strong nitric acid, and speedily carbonized by strong sulphuric acid. It acquires a blueish-grey colour by the action of iodine-water. A solution of green sulphate of iron produces no change in it; but it is darkened by the persulphate. When heated, an inflammable vapour is evolved, and a carbonaceous alkaline ash is left as a residue.

The *seeds* are small, and resemble the pips of the apple, except that they are of a lighter brown colour and are flatter.

[These seeds when wholly freed from the pulp are oleaginous and nutritive, and are employed in some parts of Africa as an article of food.—G.]

DAFFODIL.

A watery extract of this plant (Narcissus) administered to dogs was found by Orfila to cause vomiting and other symptoms of local irritation, followed by death. It acts upon the nervous system, as well as locally upon the mueous membrane of the stomach, which was found deeply reddened in some of the experiments.

ELATERIUM.

This is the dried juice of the MOMORDICA ELATERIUM obtained by pressure and subsidence. It is an irritant substance, and is well known as a drastic purgative. These properties are owing to the presence of a crystallizable principle *Elaterine*, which forms from 5 to 26 per cent. of the extract, a circumstance which is sufficient to account for the uncertainty of its effects in given doses. Elaterium is a very active substance, producing severe vomiting and purging in the dose of a grain. Even from 1-16th to 1-8th of a grain will often act violently on the bowels. Like other irritant purgatives. it may be criminally used for the purpose of procuring abortion, although it has no specific action on the uterus. The medicinal dose is from 1-16th to one-half of a grain.

ANALYSIS.—Elaterium is a pale greyish green amorphous substance, very slightly soluble in alcohol, forming a green solution. When heated on platina it leaves an alkaline ash.

The following case of poisoning by Elaterium is related by Dr. Beck. A female in Boston took a quack medicine, *i. e.* four pills, consisting of rather less than two grains and a half of elaterium and sixteen grains of rhubarb. Incessant vomiting and purging. followed, under which she sank, thirty-six hours after the last pill was taken. On dissection the mucous membrane of the stomach was found to be highly injected, the colon contracted, and all the intestines inflamed. The other viscera were healthy.

ELDER.

Dr. Christison states that the *leaves* and *flowers* of the common elder (SAMBUCUS NIGRA) act as an irritant poison, having caused in a boy severe inflammation of the bowels which lasted for eight days. (Op. cit. 607; and Ed. Med. and Sur. Jour. xxxiii. 73.) The berries of this tree do not, however, appear to possess, in the ripe state, any noxious properties. The following case of poisoning by the expressed juice of the *roots* is reported. (See Med. Gaz. xxxv. 96.) A weakly woman, 54 years of age, who had been sick all day, and thrown up a quantity of greenish matter, which she regarded as bile, was persuaded by her husband to take two tablespoonfuls of the juice of the fresh elder-root, which he himself had dug up, shaved down, and pressed. The woman soon after complained of severe pain in the abdomen. She was ordered some infusion of senna, but did not take it, as the bowels began almost immediately to act copiously. Next day the symptoms were those of enteritis, which proved fatal.

[A native species, S. Canadensis, also called Elder, is almost identical in its powers with the European plant.—G.]

The berries in a crude state excite nausea and purging.

EUPHORBIUM.

This is a gum-resin, the inspissated extract of a genus of plants which have a milky juice, and are known under the name of Euphorbia or Spurge. The extract contains nearly half its weight of an acrid resin, which acts like an irritant, producing griping pains and diarrhœa. After death the mucous membrane of the stomach and intestines is found inflamed. In one instance a teaspoonful swallowed by mistake produced burning heat in the throat and stomach, with vomiting. The individual died in three days. (Christison, 588.) It is used in veterinary medicine, and may thus occasion poisoning by mistake. The seeds and root of several varieties are equally poisonous. The following is a case of poisoning by the Euphorbium Peplus (Petty Spurge.) A boy, æt. 6, ate the plant by mistake. He was seized with vomiting and purging, spasms, small pulse, inability to swallow, insensibility, and cold extremities. He sank under these symptoms, and on inspection, the tonsils, fauces, pharynx, and larynx were found much inflamed, and containing a green-coloured mucus. The mucous membrane of the stomach, and intestines was very red, but the large intestines were healthy, with the exception of the muscular coat, which was vascular. The bladder was contracted: the lungs healthy, as well as the substance of the brain. The veins of the dura mater were distended. (Beck's Med. Jur. 832; and Med. Chir. Rev. vii. 275.) Orfila quotes what appears to be a somewhat doubtful case, in which a woman died in half an hour from about twenty-five grains of the root. (Toxicol. ii. 104.) There is no doubt that euphorbium is a very acrid substance, and that in all its forms, it possesses a strong local irritant action.

Analysis.—Euphorbium in *powder* has a light fawn colour. Strong nitric acid dissolves it, and forms a brown liquid (artificial tannin.) Strong sulphuric acid turns it of a dark reddish-brown colour. Potash gives to it a yellowish tint. Green sulphate of iron and iodine water produce in it no apparent change. Alcohol dissolves a portion (resin) which is precipitated of a milky-white when water is added. When heated on platina-wire it does not melt; but gives off a dense vapour which burns with a smoky flame, and leaves a grey residue, containing potash, lime and oxide of iron.

GAMBOGE.

It appears from experiments on animals performed by Orfila, (ii. 96.) and other toxicologists, (Wibmer, Garcinia) that this gum-resin is, in large doses, an irritant poison, but not of a very active kind. It has a peculiar taste, and when mixed with water, a very peculiar odour. The medicinal dose is from one to three or four grains. In large doses it produces pain, vomiting, with other signs of irritation in the alimentary canal, and after death the stomach is found inflamed. In small doses frequently repeated, it causes, like colocynth, hypercatharsis, attended with debility and exhaustion leading to death. The effects which it produces have been already described in speaking of colocynth (ante, page 409;) and there is no doubt that the deaths from Morison's Pills are to be ascribed to the conjoint action of these two vegetable irritants. These are the only cases in which, so far as I know, gamboge has been a cause of death in the human subject: and they must be regarded as instances of chronic poisoning.

Analysis.—Gamboge is a gum-resin of an intense yellow colour. When heated on platina it melts, and burns with a bright yellow flame, leaving a carbonaceous ash. It is soluble in water, forming an opaque solution having a slightly acid reaction,—also in alcohol and ether. The two latter menstrua may be used for separating it when it is mixed with other compounds. The alcoholic solution is precipitated by water of an opaque yellow colour, but the liquid is rendered clear and turned of a deep golden-red colour by caustic potash.

HEDGE HYSSOP.

The Gratiola Officinalis is commonly known under the name of *Hedge Hyssop*. Observations made on animals and on man, show that it is a strong local irritant when given in decoction or infusion. A series of cases observed by M. Bouvier are reported by Orfila, in four of which this plant was used, under the form of decoction, as an enema. In this state it had been prescribed for four females by some herb-doctors. The result was, that in one instance violent vomiting and purging, with syncope, were induced, and in all a strong attack of nymphomania. In another case there was constriction of the throat, with hydrophobic symptoms and convulsions. The patient died in two days. (Toxicologie, ii. 128.) The leaves of this plant might be in some instances identified botanically; but in the state of decoction or infusion, there are no tests which would determine its nature. Its active properties are owing to the presence of a principle called *Gratioline*, for an account of which see Monthly Journal of Medical Science, March 1846, 219.

[Our native G. AUREA is fully as active.—G.]

JATROPHA CURCAS.

The seeds of this plant, which grows in the West Indies, commonly known under the name of *Physic Nut*, contain a very acrid oil, which, according to the experiments of Orfila, produces burning of the throat, vomiting, inflammation of the stomach, and other symptoms of irritation. (ii. 107.) Dr. Christison states that four seeds will act upon a man like a violent cathartic; and he has known severe vomiting and purging occasioned by a few grains of the cake, left after the expression of the fixed oil from the bruised seeds. (Op. cit., 591.) Its action is something analogous to that of croton oil; but it requires, according to the same authority, twelve or fifteen drops to produce the effects of one ounce of castor oil.

It is stated by Dr. M'William, that a decoction of the leaves of this plant is used locally by the natives of the Cape Verd Islands (Boa Vista) for the purpose of exciting a secretion of milk in the female breast, and that in this way a woman who has once borne a child, and is not past child-bearing, may be on an emergency converted into a wet-nurse. (Report on the Boa Vista Fever, 1847.)

JALAP.

The powdered root of jalap (IPOMÆA JALAPA) may be given as a safe purgative to adults, in doses of from ten grains to half a drachm. Frequently repeated, or taken in a larger dose, it produces pain and violent purging. I have not met with any instance of its having proved fatal in the human subject, but it might act like colocynth and gamboge, when its use was long continued in large doses, by causing great prostration of the vital powers. Some experiments detailed by Orfila, show that this substance will cause the death of animals, by exciting violent purging, and that inflammation of the alimentary canal is among the post-mortem appearances. (Op. cit., ii. 85.) Its irritant properties are ascribed to a resinous principle, of which the powder contains about ten per cent. It is much used in medicine.

ANALYSIS.—Jalap is seen in the form of a dark reddish-brown powder. The odour is peculiar, and may serve to identify it. It is turned rapidly brown by strong nitric acid; but if adulterated with guaiacum it becomes at first green. This effect is often observed in the powder sold as jalap by druggists. The powder is immediately carbonized by strong sulphuric acid. It acquires a purple colour with solution of iodine (starch.) Caustic potash turns it of a greenish-brown colour, and green sulphate of iron gives with it a dark olive-green colour. When heated, it takes fire and burns, leaving a slight alkaline residue. *Guaiacum* powder differs from jalap in its chemical properties. It is of a brown colour, but by exposure to light it becomes green. It powerfully deoxidizes strong nitric acid, acquiring a brilliant green colour, which is rapidly changed to brown. Strong sulphuric acid produces with it a splendid crimson or blood-red compound, which changes to a dingy purple when water is added. It is highly inflammable, melts, and burns when heated with a smoky flame, leaving a carbonaceous ash. Iodine-water, and the proto, and persalts of iron, produce no effect upon it.

MANCHINEEL.

The juice of this plant (Hippomane Mancinella,) a gum-resin, is, according to the experiments of Orfila and others, possessed of strong irritant properties, owing to the presence of a volatile substance. It therefore becomes less acrid by drying, although it has been known to retain its active properties for six months. The poisonous principle exists in the fruit, bark, leaves, and roots. At first the juice has a faint taste, but this is speedily followed by a strong burning sensation, with swelling of the lips, tongue, and gums, pain in the stomach and bowels, with vomiting and liquid stools. It also acts by local application. After death the usual appearances are those of inflammation. It has been alleged that even the vapour of the tree is poisonous to those who sleep under its shade, but this is open to doubt.

It has been supposed that the negroes of the West Indies are in the habit of employing the juice of this plant for the purposes of secret poisoning; but Dr. Rufz has lately shown, by a series of elaborate investigations, that owing to its strongly irritant properties, it is not adapted for this nefarious object. He found that in animals it required three drachms of the fresh juice to produce diarrhœa, colic, and loss of appetite; but this quantity killed a strong mule in seventeen hours. An ounce, according to this gentleman, has certainly produced death in six hours; and he admits that, in doses less than a grain, it is capable of producing well-marked effects in the human subject. The sensation of burning heat and pain which it produces in the stomach, would at once make known any attempt at slow poisoning; and there is no evidence of its being an accumulative poison. (Ann. d'Hyg. 1844, ii. 213.) Dr. Rufz found that the eighth of a grain of the juice caused well-marked symptoms of irritation, with giddiness and convulsions. The poison is quite unknown in England.

Dr. Hamilton states that every part of the tree abounds in a caustic milky juice, which possesses the property of blistering the skin. The fruit resembles a small apple: it has a pleasant smell, but is intensely acrid. Dr. Hamilton ate a small fragment of the fruit immediately before breakfast. There was no inconvenience from swallowing it; but it produced a burning sensation in the stomach, which, but for the breakfast he had taken immediately afterwards, might have excited active inflammation. The milky juice produces tingling of the tongue, and excites a flow of saliva. The caustic principle of the juice is supposed to be of an alkaline nature, as its irritating properties are counteracted by lime-juice. (See Pharm. Journ., March 1846, 408.)

MEZEREON.

The bark, root, leaves, and seeds of the varieties of mezereon (Daphne) are endowed with irritant properties, owing to the presence of an acrid resinous principle. Four berries produced in a man severe hypercatharsis, vomiting, thirst, burning of the mouth and throat, with fever. (Wibmer, Daphne, 283.) Dr. Christison refers to a fatal case in a child about eight years of age, which occurred in Edinburgh, and to three others in which violent vomiting and purging (in one with narcotic symptoms) took place as a result of eating the berries; but the children recovered. (Op. cit., 601.) A case somewhat similar to these has been communicated to me by Mr. Tubbs, in which a child aged four years, swallowed some of the berries at four o'clock in the afternoon. The child, when seen soon afterwards by Mr. Tubbs, was drowsy, and the pupils were dilated. The stomach-pump was used, and five! of the berries were brought away. An emetic of sulphate of zinc and a dose of castor oil, were then given. On the day following, the child had perfectly recovered. The following is a recently reported instance of poisoning by these berries. It appears that in Germany five or six berries are often employed as a domestic purgative. But, on account of obstinate constipation, a peasant took forty. Soon afterwards he was attacked with severe pain in the abdomen, frequent and violent vomiting, and profuse diarrhœa, the stools being mixed with blood. There was giddiness, with dilated pupils and imperfect vision; extreme thirst, burning pain in the throat, extending to the stomach, and violent colic. The voice was tremulous, respiration short and difficult, pulse frequent and irregular, extremities cold, and the skin covered with a clammy perspiration. Appropriate remedies were applied, but at the time of the report, the recovery of the patient was very doubtful. The powder of the bark appears to be as active as the berries; and the decoction is also poisonous. The flowers appear to be poisonous, for, according to Gmelin, bees instinctively avoid them. Medicinally used, the active principle of mezereon bark appears to have an action on the kidneys like cantharidine.

LEATHER WOOD.

[The Dirca Palustris, a native plant belonging to the same order as Daphne is equally active. The berries are emetic and poisonous, and the fresh bark is a powerful rubefacient when applied to the skin, and when taken internally is capable of inducing violent and even fatal inflammation.—G.]

MUSTARD.

The two varieties (Sinapis nigra et albà) of black and white mustard-seed possess when powdered, strong irritant properties owing to the production by the action of water of an acrid volatile oil. The black seed is more irritant than the white. The seeds of the white mustard were formerly taken in large quantities as an aperient, but this absurd practice, as Dr. Pereira has justly observed, was attended with some danger, owing to the tendency of all insoluble substances of this description, to collect in the appendix vermiformis cœci, and there give rise to inflammation and ulceration,—changes to which this portion of the intestinal canal is especially liable. Common mustard powder may be usefully employed in most cases of poisoning as an emetic, in doses of from a tea-spoonful to a table-spoonful in from four to six ounces of water. In still larger doses it may give rise to vomiting, purging, and inflammation of the bowels.

Analysis.—The physical properties of common mustard powder are sufficient to identify it. A decoction or infusion of bruised mustard-seed possesses chemical properties which unless pointed out might lead to error in analysis. Strong nitric acid added to it strikes a deep red colour like that produced by morphia: iodic acid is decomposed by it and iodine is set free: if starch be added, the liquid becomes blue. This property is similar to that possessed by morphia. A persalt of iron (colourless persulphate) strikes a deep red colour in the decoction like that produced by meconic acid: it differs from it, however, in being immediately discharged by a few drops of a solution of corrosive sublimate.

The above effects are very similar to those produced by a solution of opium with the same reagents: there is, however, no odour of this drug. They arise from the production of sulphocyanic acid in the mustard. If a few grains of pure zinc and sulphuric acid be added to the liquid coloured by the persalt of iron,—in the case of mustard (sulphocyanogen) the red colour is speedily destroyed and sulphuretted hydrogen is evolved, as indicated by its action on paper dipped in a salt of lead. With meconic acid (opium) there is no such effect. I have not found the red colour to be thus destroyed and sulphuretted hydrogen evolved. This test was proposed by Dr. Percy: and it is applicable to those cases where sulphocyanogen is present, and creates ambiguity with the iron-test, provided the zinc employed be free from sulphur.

RANUNCULUS.

There are many varieties of this common plant, the recent juice of which, especially that of the roots, has a hot acrid taste, and is capable of acting as an irritant poison. The acridity is lost by drying. The most poisonous of the genus are the R. Bulbosus Sceleratus and Acris (Water Crowfoot.) There is no instance of their having operated fatally on the human subject, but their effects on animals, leave no doubt of their possessing poisonous properties (Orfila, ii. 130: Wibmer, Art. Ranunculus.) The following case of poisoning by the Ranunculus bulbosus is of recent occurrence. A young lady, æt. 15, ate several stems and flowers of the plant, and chewed many more, sucking the juice. In six hours she complained of a sense of heat in the throat, and sickness. These symptoms were followed by tenderness of the abdomen, delirium, and stupor, these lasted eight days, leaving her in a state of debility. She recovered under a farinaceous diet and the use of oleaginous clysters. (Med. Gaz. xxxvii. 1060.) The Ranunculus acris is popularly known under the name of Butter-cups.

SAVIN.

This, which is the Juniperus Sabina of botanists, is a well-known plant, the leaves or tops of which contain an irritant poison in the form of an acrid volatile oil of a peculiar terebinthinate odour. They exert an irritant action, both in the state of infusion and powder. They yield by distillation about three per cent. by weight of a light yellow oil, on which the irritant properties of the plant depend. The powder is sometimes used in medicine in a dose of from five to twenty grains. Savin is not often taken as a poison for the specific purpose of destroying life; but this is occasionally an indirect result of its use, as a popular means for procuring abortion, and it therefore demands the attention of the medical jurist. From cases which have been referred to me, I believe that poisoning by it is much more frequent than is commonly supposed. From the little that is known of its effects, it acts by producing violent pain in the abdomen, vomiting and strangury. Purging is not so common an effect as with other irritants. Salivation is sometimes present. After death, the œsophagus, stomach and viscera, with the kidneys, have been found either much inflamed or highly congested. There is no proof of its having any action as an abortive, except like other irritants, by causing a violent shock to the system, under which the uterus may expel its contents. Such a result can never be obtained without placing in jeopardy the life of the woman; and where abortion follows, she generally falls a victim. On the other hand, the female may be killed by the poison without abortion ensuing. Out of four fatal cases of the administration of savin and other drugs for the purpose of procuring abortion, the mother died undelivered in three, and in the fourth instance, the child died after it was born. When the vomiting and purging are very severe, abortion may be expected to follow.

The strong local irritant properties of the leaves, which depend on the essential oil, are well known, from the uses of savin ointment in pharmacy. The plant grows extensively in country places, and is easily accessible to the evil-disposed. It does not appear to have attracted much notice on the continent, for Orfila is silent on the subject, except in so far as it affects dogs. Two cases of its fatal effects in the human female were communicated to Dr. Christison. In one, a dose of the strong infusion was twice taken by a female for inducing abortion. She suffered from severe pain and strangury, aborted, and died five days afterwards. On inspection there was extensive peritoneal inflammation, with the effusion of fibrinous flakes; the inside of the stomach was red, with patches of florid extravasation. The contents had a green colour, and savin was proved to be present by the microscope. In the second, a girl was seized with violent colicky pains, vomiting, tenesmus, dysuria, and fever. After suffering several days, she died. The stomach and intestines were inflamed; the former in parts black, and at the lower curvature perforated. A greenish powder was also found in this case, and when washed and dried, it had the pungent taste of savin.

Although it is not considered that savin has a direct tendency to produce abortion, it appears, from its therapeutic employment in chlorosis and amennorrhœa, to affect the uterus. The dried powder, which, owing to the loss of volatile oil, is less energetic than the fresh tops, is given in doses of from five to fifteen grains. The medicinal dose of the essential oil is commonly from two to six drops. The infusion and decoction, which are sometimes used for the expulsion of worms, are less energetic than the fresh tops, because they cannot be prepared without giving rise to a loss of the volatile oil. The oil is not so irritant as it is commonly supposed to be; but in those cases in which it has been said to produce no marked effects in large doses, it is very probable that it was much adulterated.

A well-marked case of poisoning by the tops of savin was referred to me for examination, by Mr. Lord, of Hampstead, in May 1845. The deceased, a healthy female, had reached about the seventh month of pregnancy. She was very well on the Friday, but was seized with vomiting on the Saturday: she stated that she had taken nothing to produce it. The vomiting continued throughout Sunday, and was of a green colour. She was first seen by a medical man on Sunday evening. The symptoms were those of gastro-enteritis—great anxiety, and pulse 150. The green colour of the vomited matter was thought to be owing to bile. The vomiting appears to have continued at intervals, but it does not seem that there was any violent purging. Labour came on on Wednesday. The child was born living, but soon died: the female died on the Thursday, *i. e.* five days after having taken the poison, for there was no proof that any savin could have been taken after Saturday. On inspection, the brain was healthy, the lungs were healthy, except that the air-tubes had a dark red colour, the heart flabby: blood generally fluid. The lining membrane of the œsophagus was reddened, and had on it ecchymosed patches. One-half of the mucous membrane, from the cardiac orifice upwards, presented a dark red arborescent injection, with slight patches of ecchymosis: there was no erosion or ulceration. In the stomach a large patch of redness, about three inches in length, extended from the greater curvature towards the pylorus. The vessels of the mucous membrane were considerably injected, forming infiltrated patches especially about the lesser curvature, extending towards the cardiac end, but there was no ulceration or erosion. The stomach contained nearly eight ounces of a greenish fluid, of the appearance and consistency of green pea-soup. By examining a portion of the washed vegetable substance under a powerful microscope, and by drying a portion, rubbing it, and observing the odour, clear evidence was obtained that the green colour was owing to the diffusion of finely triturated savin-powder. The interior of the duodenum, especially towards the pylorus, was intensely inflamed, being of the colour of cinnabar. Patches of inflammation were found throughout the other portions of the canal. There was some peritonitis, chiefly of the upper part of the intestines and omentum. The kidneys were inflamed, and of a dark red colour—the bladder healthy. Green mucous matter containing savin, was found in the duodenum, but not in the lower part of the intestines. (Med. Gaz. xxxvi. p. 646.) The quantity of poison taken by the deceased could not be ascertained, but it must have been large. I estimated the quantity remaining in the stomach after five days, under frequent vomiting, at from twenty-five to thirty grains.

In a case which occurred to Mr. Newth, the patient, a pregnant female, eight hours after she had taken savin, was found lying on her back perfectly insensible, and breathing stertorously. She had been suddenly seized with vomiting, and this continued for some time. At first the case was thought to be one of puerperal convulsions. Labour came on, and she died in about four hours, during a fit of pain. She appeared to be between the seventh and eighth month of pregnancy, and the child was born dead. On inspection, twenty-four hours after death, the brain was found gorged with black fluid blood. The stomach was paler than usual, excepting in one or two spots, which were red as if blood had been effused into the mucous tissue. It contained four ounces of an acid liquid of a brownish-green colour. This, on distillation, yielded an opaque liquid, from which a few drops of a yellow oil were separated by means of ether. Some sediment found in a bottle presented, under the microscope, the characters of powdered savin. (Lancet, June 14, 1845, 677.) There can be no doubt that this was the cause of death. The action of the poison appears to have been, in the first instance, like that of an irritant, and just before death like that of a narcotic.

ANALYSIS.—When the poison has been taken in the form of decoction or infusion, no test can be applied. The fact of poisoning can then only be elucidated by the symptoms and by circumstantial evidence. If the oil has been taken, it may be separated by distillation, and obtained by agitating the distilled product with one-third of its bulk of ether. Perhaps the most common case is that where the *powder* has been taken. It will be remarked from a case reported by Dr. Christison, and from that which occurred to Mr. Lord, that in spite of great vomiting, the powder remained in the stomach for a period of five days. The contents appear like green-pea soup. That the colour is not owing to bile may be proved by diluting a portion with water, when the green chlorophylle, from its insolubility, will subside in a dense insoluble stratum, whereas if the colour were due to altered bile, the whole of the liquid would remain coloured. By washing the green matter in water, and drying it on plates of glass or mica, evidence may be obtained under a good microscope, by the rectilinear course of the fibres and the turpentine-cells, that the substance belongs to the fir tribe. The only other poison of the coniferous order is the yew (*Taxus baccata*,) but this differs from savin in having a lancet-shaped termination to the top of the leaves, while savin has a sharply acuminated point. A portion of the green powder dried and well rubbed will give the peculiar odour of savin. When freed from organic matter, it will yield, by distillation with water, the essential oil of savin.

OIL OF SAVIN.—This oil is of a light yellow colour, and it has a powerful terebinthinate odour, sufficiently peculiar to render this an easy means of identification. A greasy stain made by this oil on paper is entirely dissipated by heat, or only a slight trace of resin is left. It is lighter than water, but insoluble in it, giving to it, however, its odour and an acrid reaction. It forms a milky solution with rectified spirit, but a clear transparent solution with ether. It is exceedingly soluble in ether, and by this menstruum it may be separated from watery liquids as the ether floats with it to the top. Nitric acid in the cold, slowly gives to the oil a dark red-brown colour.

[Many cases have occurred in the United States, where this oil has caused death, being taken for the purpose of inducing abortion.—G.]

SCAMMONY.

This substance, from the Convolvulus scammonia, which is much used in medicine, is capable of producing, in large doses, great irritation of the alimentary canal. The medicinal dose is from ten to twenty grains: in larger doses its principal effect is to produce hypercatharsis, and to operate injuriously like gamboge and jalap, although it is considered not to be so energetic as either of these substances.

Analysis.—Scammony is usually seen under the form of a dark grey *powder*. Nitric acid turns it immediately brown. Sulphuric acid carbonizes it on contact. Iodine-water acquires with it slowly a deep purple colour. Potash dissolves it in part, and acquires a deep greenish-brown colour. A solution of green sulphate of iron produces with it no marked change; a solution of persulphate is slowly darkened. When heated on platina, it takes fire and burns with a smoky flame, leaving a grey alkaline ash.

Ipecacuanha is not very likely to be mistaken for scammony: nevertheless it will be proper to state the results of some experiments on this powdered root. Ipecacuanha in powder has a fawn-brown colour: by strong nitric acid it is turned of a rich green colour, passing speedily to brown. Sulphuric acid carbonizes it on contact. Iodine-water gives to it a deep blue colour. Potash has no immediate effect, but the liquid becomes slowly brown. A solution of green sulphate of iron produces slowly in the mixture a deep greenish colour: the persulphate is speedily darkened. When heated on platina it burns without melting, and leaves a white ash. *Contrajerva* powder strikes a blue colour with strong nitric acid, before passing to a brown.

SORREL.

There are two plants known as sorrel—the *Rumex acetosa* (common sorrel,) and the *Oxalis acetosella* (wood-sorrel.) Both of these plants contain binoxalate of potash, a salt the poisonous properties of which have been already described (ante, p. 228.) The quantity present, however, is small, and it appears to be smaller in the common sorrel than in the wood-sorrel. Orfila states that the proportion is about 1-500th part by weight, and he considers that the presence of this in soup or other articles of food, may form a serious objection to the processes for detecting oxalic acid in cases of poisoning. (Toxicologie, i. 190.) According to Mitscherlich, 100 pounds of wood-sorrel yield 20 pounds of juice, and from this three-quarters of a pound of binoxalate may be obtained. (Lehrbuch der Chemie, ii. B. s. 37.)

Toxicologists have not enumerated these plants among vegetable poisons: they have been commonly treated as pot-herbs. Wibmer states that they have a slightly irritant action on the stomach. Mr. Hanks has reported two cases, in one of which very serious symptoms were induced in a child who had eaten common sorrel (Rumex acetosa.) A child, ætat. 6, suddenly lost his appetite, complained of sickness and heaviness in the head, and soon afterwards fainted. When he recovered he was unable to stand, and vomited a quantity of greenish-coloured matter. Insensibility came on, with convulsions of the extremities. The cause of his illness was not then suspected, and the patient continued to suffer for several days, complaining of soreness of the epigastrium, and pain extending from the fauces to the stomach. There was also great thirst, and he occasionally vomited green vegetable matter. He recovered under treatment in about ten days.

In the second case the patient suffered chiefly from severe pain in the bowels. The symptoms were soon relieved by the action of an emetic—which in this, as in all other cases of vegetable irritant poisoning, is the appropriate remedy. (Med. Gaz. vol. xl. page 69.)

It appears somewhat difficult to refer these effects to the small quantity of binoxalate of potash present in these plants, yet, as in other instances, the recent vegetable may have a more powerful action than the quantity of the poisonous salt actually contained in it, would indicate. In the first of the two cases it was remarked by Mr. Hanks that four leeches which were applied to the skin, dropped off dead. A similar fact has been observed in poisoning by oxalic acid. (p. 218, ante.) This gentleman refers to a case of recent occurrence in Bath, in which the plant proved fatal to a child. Sorrel was found in its stomach, the lining membrane of which was injected and diffusely tinged.

Analysis.—The leaves and shoots of these plants, admit of identification only by their botanical characters. If the quantity eaten be large, binoxalate of potash may be separated from the contents of the stomach by making a decoction. This must be filtered hot, as six-sevenths of the salt are precipitated from a hot solution by cooling. (For the chemical tests, see Binoxalate of Potash, ante, page 228.)

STAVESACRE.

This plant is known under the name of Delphinium Staphysagria, or Palmated Larkspur. Its seeds are acrid, and produce the usual symptoms of irritant poisoning. (Orfila, Op. cit. ii. 121.) Their poisonous properties are owing to the presence of an alkaloid known under the name of Delphinia.

Analysis.—Stavesacre-seeds are of an irregular triangular shape—of a dark colour, and have an irregular surface. They are convex on one side and concave on the other, and are about a quarter of an inch in length. Nitric acid poured on the bruised seed brings out a faint reddish colour. Strong sulphuric acid produces a beautiful pink, passing to a deep pink-red colour. Iodine water gives a deep red-brown colour, and caustic potash a dark olive-green. When heated on platina the seed burns with an intensely bright smoky flame, leaving a skeleton of the seed as a grey alkaline ash.

Delphinia.—This is the solid uncrystallizable alkaloid obtained from the seeds. It is seen in the form of a light-brown powder. It is scarcely dissolved by water at any temperature, but is easily dissolved by alcohol and ether; also by diluted acids. It possesses a strong acrid persistent taste. It melts at 248°, and at a little higher temperature it burns and is decomposed. When concentrated nitric acid is poured onit, it becomes at first of an ochreous, and afterwards of a dingy red colour. Strong sulphuric acid produces with it an intense blood-red colour, which becomes heightened by the addition of powdered black oxide of manganese.

In addition to the plants just considered, irritant properties are assigned to the juices of the following:—Caltha Palustris (Marsh Marigold,) Clematis Vitalba (Virgin's Bower,) Cochlearia Armoracia (Horseradish,) Cyclamen Europæum, Fritillaria Imperialis, Lobelia Syphilitica, Plumbago Europæa, Rhus Toxicodendron and Radicans, Rhododendron Chrysanthum, Sedum Acre (the common Stone crop.) They cannot be regarded, however, as active poisons.

[Besides those enumerated by the author, the following native plants are active and in some instances have produced poisonous symptoms. Sanguinaria canadensis, Melia azederauch, Spigelia marilandica, Symplocarpus fætidus,

Podophyllum peltatum, Euphorbia corollata, Phytolacca decandra, Celastrus scandens, the different species of **Apocynum**, &c.—G.]

CHAPTER XXXII.

VEGETABLE IRRITANTS CONTINUED—CASTOR-SEEDS—CREASOTE—CROTEN OIL AND SEEDS—OIL OF TAR—OIL OF TURPENTINE—PYROXYLIC SPIRIT—DIPPEL'S OIL—FUSEL OIL—DECAYED AND DISEASED VEGETABLE MATTER—EFFECTS PRODUCED BY DECAYED CARROTS—POTATOES—MOULDY BREAD—POISONOUS VEGETABLES IN BREAD—POISONOUS FLOUR—POISONOUS GRAIN—EFFECTS PRODUCED BY ERGOT OF RYE.

CASTOR SEEDS.

Of castor oil itself nothing need be said. It is not commonly known that the seeds of the plant (Ricinus Communis) from which this oil is extracted, contain in the embryo and seed-coats a very active poison, and that a few of them are sufficient to produce violent purging and death. The following is an instance of poisoning by these seeds, the only one with which I have met. The deceased, aged eighteen, was the sister of a gentleman who was at the time attending my lectures at Guy's Hospital. The deceased, it appears, ate about twenty castor-oil seeds; one of her sisters ate four or five, and another, two. This was on a Wednesday evening. In the night, they were all taken ill. About five hours after the seeds were eaten, the deceased felt faint and sick; vomiting and purging came on, and continued through the night. On the following morning, she appeared like one affected with malignant cholera. The skin was cold and dark-coloured, the features pinched and contracted, and the breath cold; the pulse was small and wiry; there was restlessness, thirst, pain in the abdomen, and she lay in a sort of drowsy, half-conscious state. Whatever liquid was taken was immediately rejected, and the matters passed by stool, consisted chiefly of a serous fluid tinged with blood. She died in five days without rallying; the two other sisters recovered. On inspection, a very large portion of the mucous membrane of the stomach was found abraded and softened in the course of the greater curvature. There was general vascularity of the organ, and the abraded portion presented the appearance of a granulating surface of a pale rose-colour; it was covered by a considerable quantity of slimy mucus. The small intestines were inflamed, and the inner surface of them was abraded.

The irritant poison resides in the embryo, and is not expressed with the fixed oil, which is a mild purgative, unless a high temperature be employed, when the oil obtained is observed to possess much more drastic properties. The effects produced on the sisters who recovered, bear out the general opinion, that two or three of these seeds will operate as a violent cathartic. Wibmer quotes a case from Bergius, from which it would appear that even one seed without the skin, will produce severe vomiting and purging. (Arzneimittel. ii. 413.)

Analysis.—These seeds may be identified by the peculiar variegated character of the exterior.

CREASOTE.

This is an oily-looking liquid, obtained by a complex process from the oil of tar. It is volatile, and has a very powerful odour, resembling that of smoked meat, which is sufficient to identify it even when it is present in very minute proportion. In its ordinary state, it is caustic or corrosive, its chemical action being due to its strong tendency to combine with and coagulate albumen. When taken in a large dose, it produces a hot and burning sensation in the mouth and fauces; and this is followed by nausea, vomiting and purging,—the symptoms sometimes resembling those of dysentery. It exerts in some instances a remote action on the urinary organs, giving rise to strangury. The post-mortem appearances caused by a fatal dose, would doubtless be similar to those met with in the action of other irritants. The medicinal dose is one or two drops diffused in water, and gradually increased. In one case forty drops were given with impunity, and in another instance ninety drops were administered in half a day, without any bad symptom. (Pereira, Mat. Med. i. 421.) Probably the dangerous symptoms would depend less on the quantity, than on the degree of concentration. In the event of this substance being taken as a poison, albumen should be freely administered.

Analysis.—Its odour is sufficient to identify it. When heated in a platina capsule it takes fire and burns with a thick smoky flame, leaving no residue. In a concentrated state, it coagulates albumen. When pure it is perfectly neutral, almost colourless, and of oily consistency.

CROTON SEEDS AND OIL.

The Croton-seeds owe their poisonous properties to the presence of an acrid oil. One or two grains of the *seeds*, when swallowed, are sufficient to produce severe pain in the abdomen, with copious watery motions. Even the dust of the seeds, when inhaled, has caused alarming symptoms. Dr. Pereira mentions the following case:—A man had been occupied eight hours in emptying packages of the seeds, and had thus been exposed to the dust. He first experienced a burning sensation in the nose and mouth, tightness in the chest, lachrymation, and pain in the epigastrium. He then became giddy, and fell down insensible. When admitted into the London Hospital the man was in a state of collapse, complained of burning pain in the stomach, throat and head, and of swelling and numbness of the tongue. The epigastrium felt hot and tense, the pupils were dilated, the breathing short and hurried, pulse 85, and the surface cold. He complained of pain in the epigastrium for several days; but it is singular that there was no diarrhœa. (Mat. Med. ii. 1114.)

The oil has a hot burning taste. One or two drops are commonly sufficient to produce pain in the abdomen, and diarrhœa; but Dr. Traill states that a female patient usually took three drops for a dose without inconvenience. (Outlines, 149.) In large doses, the pain becomes hot and burning, extending from the mouth downwards; there is violent vomiting with purging, and the patient sinks exhausted. After death, the alimentary canal is found inflamed. Even the endermic application of the oil is stated in some cases to have produced severe symptoms, although, according to Dr. Buchanan, it acts only as a local irritant. (Medical Gazette, xxxix. 671.) A case occurred in Paris in 1839, where a man swallowed by mistake two drachms and a half of croton oil. In three quarters of an hour, the surface was cold and clammy, the pulse imperceptible, respiration difficult, and the extremities and face were as blue as in the collapsed stage of cholera. In an hour and a half diarrhœa set in; the stools were passed involuntarily, and the abdomen was very sensitive to the touch. The patient complained of a burning pain in the course of the œsophagus. He died in four hours after swallowing the poison; and it is singular that there was no marked change in the mucous membrane of the stomach. (Orfila. Tox. i. 108.) The poisonous properties of croton oil are owing to a fatty acid (crotonic acid,) which it contains in uncertain quantity. Probably this may explain why from six to ten drops of the oil may be sometimes given without causing much purging. It commonly begins to act speedily, *i. e.* within half an hour. The medicinal dose of it is from one to three drops.

This oil acts as a poison on animals. Many instances of its effects on animals have been collected by Wibmer. (Arzneimittel. i. 215.) A curious case in reference to its effects on the horse, came to trial a few years since. A veterinary surgeon administered, as a medicine, fifteen drops to a horse. The lips of the animal became swollen, and the skin peeled off; the horse suffered evidently great pain, and after lingering a short time, died. An action was brought by the owner of the horse at the Oxford Aut. Cir. 1838, for the recovery of its value. From the evidence then given, it seemed probable that the animal had really died from a very small dose of the oil, although there was reason to believe that a much larger quantity was given, than was here alleged to have caused death. Wibmer mentions two instances in which twenty and thirty drops were given to horses without materially affecting them.

Analysis.—Croton oil is a fixed oil of a lemon-yellow colour. It has a peculiarly unpleasant odour, and a hot acrid burning taste. It has a faint acrid reaction, which it imparts to water; and as it is of lower specific gravity, it floats on the surface. It is very soluble in ether, and by this liquid it may be separated from organic liquids. When warmed with nitric acid, the oil is turned of a dark-brown colour: and there is an abundant evolution of nitrous acid vapour.

Croton *seeds* are of an oval form, and about three-eighths of an inch in length. They are covered with a dusky thin blueish-coloured brittle shell, having within a yellowish-white oleaginous and easily sectile kernel, which forms the great bulk of the seed. When boiled in a solution of potash holding dissolved some oxide of lead, they are blackened, thus indicating the presence of sulphur. Like all the varieties of vegeto-albumen, the kernel is turned of a deep red-brown colour, when it is boiled in concentrated muriatic acid.

OIL OF TAR.

This is a powerful vegetable irritant. In 1832, about ten drachms of it caused the death of a gentleman, to whom it had been sent by mistake for a black draught. The party who sent it, was tried for manslaughter, but acquitted.

This liquid appears to be somewhat uncertain in its operation. Dr. Christison quotes a case in which *four ounces* taken by an adult in mistake produced pain and violent vomiting. The man speedily recovered! (On Poisons, 967.) In another instance, two or three draughts of this liquid proved fatal to a boy in twenty-four hours. The pulmonary mucous membrane was found much injected, but it is remarkable that the stomach and intestines were natural. Whether swallowed or employed as an enema, this substance is likely to exert a powerfully local irritant action.

Analysis.—Oil of Tar is a thick treacly-looking liquid, of a dark-brown colour. It has a strong and peculiar odour, is insoluble in water, but heavier than this liquid, giving to it its odour and taste. It is dissolved by alcohol, but is again precipitated milky-white by water. It is highly inflammable, and burns with a thick carbonaceous flame.

OIL OF TURPENTINE.

This very common liquid, which is so easily identified by its powerful odour, does not appear to exert any strong action as an irritant poison. It is often given with impunity in large doses to young children as a vermifuge. In the following case, reported by Dr. Evans, an infant, æt. fourteen months, swallowed *four ounces* by mistake, and recovered. The child was found two hours after the occurrence in a comatose state; pulse 130; tunica conjunctiva injected; pupils dilated; eyes watery; face flushed; breathing hurried, strangury; urine the smell of violets; bowels painful, particularly along the course of the spermatic vessels. He was ordered an emetic of ipecacuanha. Vomiting was soon excited, and briskly kept up by tepid water. The contents of the stomach had a strong odour of turpentine. After the operation of the emetic, cold was applied to the head, and flannel cloths wrung out of hot water, to the epigastrium. At 6 P. M., ten hours after the accident, he was much improved; was quite lively; pulse 120; bowels loose; had passed eight small worms. On the following day he was decidedly better; slept well during the night; slight pain in the bowels on pressure. Castor-oil was given. From this time, he improved daily, suffering only from a little excitement about the brain; and in four or five days he had perfectly recovered. (Brit. Amer. Journ. of Med. and Phys. Science, Nov. 1846.) The treatment contributed to recovery in this case. When this poison has been swallowed, it will be indicated by the odour of the breath and urine.

Although I believe there is no case on record of the destruction of life by oil of turpentine, it may excite a violent irritating action in the kidneys, tending to strangury. It may also cause hypercatharisis. The oil can hardly be called a poison, yet it may in some instances seriously affect the constitution.

ANALYSIS.—Oil of Turpentine would be sufficiently identified by its odour and inflammability. The fact of poisoning by it, would be indicated by the odour in the breath and urine.

Another kind of Turpentine, the BALSAM OF COPAIBA, has been known to cause serious symptoms. Half an ounce was administered to an adult as an enema. This was soon followed by pain in the stomach, vomiting, and general uneasiness. The man had convulsions, and for three days he was unable to speak. He slowly recovered. (Brit. and For. Med. Rev. xvii. Jan. 1840, 268.)

PYROXYLIC SPIRIT. (NAPHTHA.)

PYROXYLIC SPIRIT, also called PYROLIGNEOUS ETHER, is a rectified product of the destructive distillation of wood. It is vulgarly known and sold under the name of NAPHTHA, although it entirely differs from this compound in chemical constitution and properties. But little is known concerning its operation on the system:—it appears, however, to be analogous to that of Oil of Turpentine. Large doses produce nausea, colicky pains, and vomiting; hence it may be regarded as an irritant. It has been suggested that it might be taken by intemperate persons as a substitute for alcohol, and a case of this kind, in which it was supposed to have caused death, was once referred to me: but it was probable that the individual had died from natural causes. Common naphtha always contains tar, and the presence of this would, of course, increase its irritant properties.

ANALYSIS.—It is a yellow liquid, of a very offensive odour, and hot pungent disagreeable taste. It possesses an acid reaction, is highly inflammable, burning with a smoky flame, mixes readily with alcohol in all proportions, but is rendered milky on the addition of water, owing to the separation of tarry matter. Some of these properties are modified by its rectification. Its odour at once distinguishes it from alcohol, and would enable us to detect it in the dead body. Some years since, it was sold for commercial purposes largely mixed with alcohol.

DIPPEL'S OIL.

It may be here the place to notice the properties of some other oleaginous compounds which are products of decomposition.

Dippel's Oil must be regarded as the empyrematic product of the distillation of nitrogenous matter. In the crude state it has been given as a poison, and its action appears to be similar to that of oil of tar. Its strongly irritant properties are probably dependent on the admixture of tarry matter. It is easily accessible to the public, and may thus be taken or administered in mistake for other liquids.

Analysis.—Dippel's oil, as it is in the first instance procured by the destructive distillation of nitrogenous matter, is of a pale yellow colour, but after a time it becomes darker and thicker, resembling tar. When redistilled with water, it is nearly colourless, of a penetrating ammoniacal odour, but it becomes brown by exposure to air. It has an alkaline reaction, is soluble in water, alcohol, and alkalies. Like oil of turpentine, it causes a violent decomposition of strong nitric acid.

FUSEL OIL.

Our knowledge of the poisonous properties of this liquid, which is a product of the vinous fermentation, is derived from the researches of Dr. Furst of Berlin. It does not appear to be a very energetic poison. Two drachms thrown into the stomach of a rabbit caused great restlessness and loss of muscular power, but the animal soon recovered. A similar quantity killed another rabbit in about two hours: the principal symptoms were depression and difficulty of breathing. On inspection, there was extravasation of dark brown blood at the cardiac end of the stomach, and the mucous membrane presented brownish-red points. The duodenum and upper part of the jejunum were reddened and filled with a white mucus, the kidneys healthy and bloodless, the lungs somewhat redder than natural. Three drachms killed a rabbit within an hour. Half an ounce caused death in a quarter of an hour, and one ounce in four minutes. Fusel oil appears from these experiments, to have in the first instance a stimulating and afterwards a depressing action. In small quantities, it produces intoxication. I have experienced the effects of the vapour, and found them to be giddiness, accompanied with a feeling of suffocation and a sense of falling. Headach followed, which lasted for half an hour. Fusel oil is absorbed, and the odour may after a time be easily recognised in the breath. (Med. Gaz. xxxv. p. 430.)

Analysis.—Fusel oil is a colourless oily fluid, lighter than water, of a very offensive odour; it has a sharp burning taste,—and burns with a brilliant flame. It coagulates albumen and casein, and appears to soften and dissolve the lining membrane of the alimentary canal. It is a ternary compound of oxygen, hydrogen, and carbon.

DECAYED AND DISEASED VEGETABLE MATTER.

Vegetable matter, when eaten in a state of decay, is capable of exciting pain, vomiting, purging, and other symptoms of poisoning. Potatoes, carrots, turnips, and other esculent vegetables, in a state of decomposition, may thus excite serious symptoms, which might be referred to mineral poison.

DECAYED CARROTS.

The following case of poisoning by *decayed carrots* has been reported by Dr. Lieber. A girl, eight years old, who had gone to bed cheerful and well, on awaking complained of headach. Violent vomiting came on, which was quieted by camomile tea, but convulsions were soon after observed. They continued till noon, when Dr. Lieber was sent for, but he did not see her for three hours. She was then lying, without consciousness, on her back, and breathing feebly; her face was rather puffy; her pulse quiet and full; the bowels not open. Leeches to the head and a clyster were prescribed, but before these remedies could be used, the girl was dead. No cause for the illness could be discovered, except her having eaten a considerable quantity of *half-rotten carrots*, which had been thrown into the court-yard. A boy belonging to another family, who had eaten of these carrots, was also ill, but recovered after spontaneous vomiting and diarrhœa. A post-mortem examination, twenty-eight hours after death, confirmed Dr. Lieber's conjecture as to the cause.

The membranes of the brain were very full of blood; the brain, which was normal in consistence, was less so; the sinuses, were distended with blood. There was no effusion in the ventricles, and the *plexus choroides* were rather paler than usual. There was no disease in the cavity of the thorax. The external surface of the stomach was unaltered, but on the internal coat, particularly at the greater curvature, there were several inflamed spots of different sizes. They were of a dark-red colour,—the finest vascular ramifications could be distinctly seen, and the colour could not be washed off with water. Blueish spots were perceived in the intestines, but only in their peritoneal covering; the mucous membrane bore no marks of inflammation. (Med. Gaz. xxxi. 270.)

DECAYED POTATOES.

Dr. O'Brien has reported five cases, from which it would appear that potatoes in a diseased or decayed condition, are capable of exciting gastro-enteritis. The symptoms in one case were, a quick pulse, white tongue, dry harsh skin, and obstinate constipation. In forty-eight hours, the face presented an erysipelatous appearance, and there was great nausea, with tenderness in the epigastric and right hypochondriac regions. There was also restlessness, with excruciating pains in the muscles and in the joints. All the patients recovered. (Dublin Hospital Gazette, Feb. 1, 1846, p. 184.) In a series of cases recorded by Dr. Peddie, where a family had subsisted six weeks on refuse-potatoes picked up on the surface of fields, the symptoms were more severe, and in two instances death ensued. The potatoes were of a green and of a deep purple colour, and had an exceedingly bitter and disagreeble taste, so much so that no mode of preparation rendered them palatable to the destitute family which suffered from their effects. In a very few days after using them, the whole family were seized with severe griping pains in the bowels, followed by diarrhœa of a green watery kind. These symptoms continued with short intermissions, during the whole of the time that the potatoes were used for food. Two of the children died. (Ed. Med. and Surg. Journ. xxxix. p. 384.) The following is a more recent instance of the injurious effects of potatoes. A woman, aged forty-three, who had no previous ailment, was seized with severe colic, vomiting, and purging, the stools consisting of fragments of potatoes floating in a mucus-like fluid; she had cramps in the calves of her legs and fingers, and her thumbs were contracted; she was very weak; had a shrivelled appearance; pupil much dilated; face, chest, and extremities cold; eyes haggard and sunk in the orbits; pulse 100, quick, small, and compressible, sometimes scarcely perceptible; tongue clean; epigastrium tender on pressure; respiration irregular, sometimes interrupted. It appeared from the account given by her parents, that she had been for half an hour without consciousness, pulse, or respiration—in fact, like one dead—before she received medical assistance; from this state she revived after discharging a large quantity of potatoes by vomiting and purging. A mixture containing five grains of tartar-emetic was ordered, of which a spoonful was directed to be given every ten minutes; a sinapism was applied to the pit of the stomach. The purging and vomiting of pieces of potatoe continued. The woman having quite regained her consciousness, stated that during the last fifteen days, she had eaten a great quantity of unripe potatoes—that she had had diarrhœa for eight days, and that at her last meal at noon, she had eaten more of them than at any previous period. At half past ten o'clock in the evening the general heat of surface returned, the cramps diminished, and the pulse became fuller and stronger; tongue continued clean; the emetic mixture was ordered to be continued. The vomiting, which had not ceased, became simply bilious. On the next day she had an opening mixture, and after this she gradually recovered. (Journal de Chimie Med. 1846, 268.)

In this instance, the effects were ascribed to the unripeness of the potatoes. It has been supposed that *Solanine* sometimes exists in potatoes, and confers on them poisonous properties, but there is no direct proof that this is the case. According to Liebig, Solanine is generated in the shoot of the potatoe when it is allowed to germinate in the dark.

MOULDY BREAD.

That universal article of food, bread, has frequently caused symptoms resembling those of poisoning, for which it is not always easy to account. In the Annales d'Hygiene, 1843 pp. 35, and 347, will be found communications on this subject from MM. Guérard, Chevallier, and Gaultier de Claubry. The changes which take place in the decomposition of flour and bread, and the production of various kinds of *mouldiness*, are here investigated, together with the effects of such bread upon the animal system. It would appear that in some parts of France, the peasantry manifest no repugnance to the eating of *mouldy bread;* and that in many instances, the practice appears to be attended with no ill effects. The nature of the mould produced, however, is subject to great variation; and it is not improbable, as M. Chevallier suggests, that in some cases a poisonous principle is actually developed. In two instances of children, who had partaken of mouldy rye-bread, symptoms resembling those of irritant poisoning supervened. The countenance was red and swollen, the tongue dry, the pulse quick; there were violent colics, with pain in the head and intense thirst. Vomiting and purging supervened, with a state of collapse; but the children eventually recovered. These symptoms were ascribed to the production of "*mucor mucedo*" in the bread. In 1829, alarming effects having followed from the use of a certain kind of bread in Paris, M. Barruel was called upon to determine whether or not, any irritant poison had become accidentally intermixed with it. The bread was simply in a mouldy state; there was no trace of the presence of any mineral or vegetable poison. These facts, together with experiments performed on animals, show that bread in a state of mouldiness, may not only produce symptoms of poisoning, but actually cause death; and as it is impossible to distinguish the noxious from the innoxious kind of mould, the use of all bread in such a condition should be avoided. The red or orange-coloured mould (*penicilium roseum*) appears to be more noxious than the greenish blue (*penicilium glaucum*). Accurate researches have shown that inferior descriptions of flour are very prone to undergo these changes when they are kept in a damp place. When the bread is first baked, it appears good and wholesome; but the crumb contains the sporules of the mould not destroyed by the heat of baking; and these only require favourable conditions (humidity) for their development. The corn of one season may be particularly prone to this change, from the weather having been unfavourable to its growth. M. Gaultier de Claubry found the corn grown in France in 1841, to undergo this noxious conversion readily (*penicilium roseum*) in the state of flour or bread; but it was not so with that grown in 1842. The season of 1841 was remarkably wet for the harvest.

Fresh Bread may occasionally produce serious effects on the body, owing to the flour becoming accidentally mixed with various descriptions of poisonous pulse or grain. Frequent use of the *Brown bread* of London, has been known to occasion vertigo, lethargy, and other unpleasant symptoms, indicative of an affection of the brain and nervous system. These symptoms have been ascribed to the accidental admixture with the corn, of the *Lolium temulentum*, or bearded darnel. In other instances, the use of it has been followed by a pricking sensation and a sense of distention in the stomach, with colic; and these symptoms have ceased only after some hours.

Rye Bread, which is largely consumed by the inhabitants of Northern Europe, but is not much used in this country, is liable to contain ergot, and to give rise to a peculiar train of symptoms. (See post, Ergot, p. 432.) Bread which contains ergot and rye-flour in equal parts, is of a brown colour outside, and is full of cracks. It is much heavier than good rye bread; when broken, it is porous, and of a greyish-blue colour. It has a sweet taste, leaving a sense of bitterness behind it. It is more liable to become moist than good bread.

Professor Henslow on one occasion found ergot in wheat which had been sent to the miller. (Pereira, ii. 915.) Those frequent diseases in wheat, smut and mildew (*Uredo frumenti*,) are not adequate to account for the occasionally injurious effects of ordinary bread. There is no proof that the diseased grain possesses any poisonous properties, even when taken in much larger quantity than it is ever likely to be found in bread; but the greater part of the rust is dissipated in the preparation of the grain. A case was referred to me lately, in which sickness, diarrhœa, and severe colicky pains, followed the use of brown bread at breakfast. The presence of poison being suspected, a portion was sent for examination, but none could be detected. There was no mouldiness: on gently shaking the bread, no sporules appeared in a shower of dust, nor had the bread undergone any change of colour. This subject is of more importance than may at first sight appear, since an attempt at poisoning may be unjustly attributed to innocent persons. A magisterial investigation took place in this city a few years since, under the following circumstances. The whole of the members of a family, eight persons, were seized with violent vomiting soon after a meal, at which they had partaken of brown bread. The symptoms somewhat resembled those of arsenic. The bread and other articles about the premises were carefully examined, but no mineral poison was found; and a minute investigation showed that the effects must have been really due to the bread, which had been procured in nearly a fresh state from the shop of a respectable baker just before it was eaten by the family. Chemistry threw no light upon the matter. The symptoms came on in a quarter of an hour; they were chiefly indicated by vomiting and tremor of the limbs. In the case of an infant there were convulsions. The effects were principally those of an irritant; but there was no purging, and all ultimately recovered.

POISONOUS FLOUR.

The following remarkable case of poisoning by flour occurred to Dr. Tait, in September 1844. Seven of the members of a family soon after dinner felt an uneasy sensation in the stomach, accompanied in some by great sickness and inability to keep the erect posture. In one there was no vomiting, but nausea and griping pain. Two, who speedily got rid of the contents of the stomach by vomiting, soon recovered. In four, vomiting supervened only after five or six hours, and it was then attended with lancinating pains in the stomach and bowels, burning heat in the epigastrium, a dry sensation in the mouth, thirst, pain in the forehead, and restlessness. This was followed by purging, depression, paleness, and collapse. In all but one young man the pulse was quick and feeble; in him it was full and bounding. He complained much of headach, and his eyes were prominent and congested. The mother suffered most severely: when first seen, her countenance was pale and collapsed, her extremities were cold, and the pulse was quick and feeble; she was conscious;—the pupils were contracted, and sensible to light;—the tongue was covered with a thick frothy mucus;—there was intense thirst, but all that was taken into the stomach, was speedily ejected. She appeared to suffer great agony immediately before vomiting, and there was excessive tenderness of the abdominal parietes. The matter thrown from the stomach consisted chiefly of mucus streaked with blood. There was also some diarrhœa. She did not recover for several weeks. In the others, the chief symptoms on the day following the accident, were headach and thirst. (North. Journ. Med., Sept. 1845.) These serious symptoms were traced to some *American flour*, which had been used at dinner for making an apple-dumpling. It was clear, from the time of occurrence and their general resemblance, that they had been caused by some article of food taken at dinner. Broth and meat were the other articles used; but these could not have caused them, because some members of the family dined solely on them, and were in no way affected. Those who took the apples and not the paste of the dumpling, were also unaffected. The mother, who suffered most, dined almost entirely upon the paste, and the violence of the symptoms was found to have a close relation to the different quantity of paste eaten by the patients. The flour, when examined, appeared perfectly good; it was analysed, but no mineral poison existed in it. It was thought that some acrid vegetable poison might be accidentally mixed with it;—but the symptoms did not bear out the suspicion that this was owing to the presence of the *Lolium temulentum*.

POISONED GRAIN.

Other kinds of pulse, such as the *Lathyrus cicera* and *Ervum ervilia* (Bitter vetch,) may occasionally render bread poisonous. In some parts of the continent, a white bread is made from the flour of the *Lathyrus;* but owing to the injurious effects resulting from it, its use has been repeatedly prohibited in several continental states. Loudon states, that when mixed in equal parts with wheaten flour, it makes a white and apparently wholesome bread; but weakness of the knees and spasmodic contractions of the limbs have followed its use. Cattle and birds which are fed on the seeds become paralysed; the seed from a strong moist soil is more injurious than that cultivated in one which is light and dry. (Encyc. of Plants, 620.) A more recent exemplification of the injury resulting from the use of the *Lathyrus* flour in bread, has been furnished by M. Vilmorin. He remarked that the use of this bread for a few weeks, produced complete paralysis of the lower extremities in a young and healthy man. Six or seven individuals of the same family, who had eaten it, suffered more or less from similar symptoms, and one had died. A physician who practised in the district, remarked that paralytic affections were very common among the poor who subsisted on this bread, while they rarely occurred among the better classes. When the *Lathyrus* flour formed only *one-twelfth* part, no inconvenience was observed to attend its use: in a proportion greater than this it became injurious; and when it amounted to one-third part, the effects might be serious. The *Lathyrus cicera* has a reddish-coloured flower; its seed is of an ash-grey colour, uniform and angular. (Ann. d'Hyg., Avril 1847, 469.) The *Ervum ervilia* or bitter vetch is a plant of the same family; it is less cultivated than the *Lathyrus*. Its seed is of a reddish-grey colour, smaller than that of the *Lathyrus*, and almost round. The flower is small, and of a dirty white hue. (The reader will find a further account of the serious effects of these seeds on man and animals, in the Ann. d'Hyg., 1841, 126.) The use of such an admixture with wheaten flower is dangerous, and should be strictly prohibited by law.

In 1844 an investigation was ordered by the French government into the condition of the flour obtained from Egyptian corn. It appeared that the grain and flour abounded in *weevil;* and the question was, whether the presence of this insect rendered it unwholesome for bread. The commission reported that some specimens were of bad quality, owing to deficiency of gluten, the presence of bran, and the ova and bodies of numerous insects; but that it was only likely to prove injurious to health by its being deficient in the nutritious principles which exists in good flour; that the presence of the weevil acts in no other injurious way, and that the bread made from the flour is not poisonous. (Ann. d'Hyg., 1846, i. 98.) The corn was, notwithstanding this opinion, condemned, but, on appeal, the judgment was reversed. (Ib. ii. 161.)

It is said to be the practice with bakers to mix alum and bone-earth with flour, to increase the weight and give whiteness. These adulterations may be determined by incinerating a dried portion of bread. Alum should not exist in good bread, but phosphate of lime, in small proportion, naturally exists in most of the cerealia. The artificially introduced phosphate might be dissolved out of the dried and powdered bread, by digesting it in diluted muriatic acid.

In some instances, whether arising from accident or design, the bread may be found really poisoned. A large portion of the population of Dresden narrowly escaped poisoning by arsenic, in consequence of partaking of bread from the shop of a baker, who had employed a rat-catcher on his premises to poison vermin. By some accident the arsenic had become mixed with the flour. (Journ. Chimie Médicale, Mars 1847, 133.) In February 1847, a portion of cake which had been placed before a family was forwarded to me for examination in consequence of certain serious symptoms having followed its use; and I found it to be impregnated with arsenic; but how or in what way the poison had been introduced into it, could not be determined. It is therefore always proper to submit the suspected bread, or other article of food, to analysis, since poison may be unexpectedly discovered in it. (On the poisonous properties of bread, see Ann. d'Hyg. 1834, ii. 179; 1835, ii. 240; 1843, i. 41, 347; Henke, Zeitschrift der S. A., 1842, ii. 185; 1844, i. 286, ii. 215.)

ERGOT OF RYE.

Some toxicologists rank this substance, known as SECALE CORNUTUM, among narcotico-acrid poisons; others exclude it from the three classes, regarding it as a poison *sui generis*. Although its effects are in some instances peculiar, yet when taken in large doses the symptoms produced very much resemble those caused by the vegetable irritants. Without assuming that it strictly belongs to this class of poisons, it appears to me that it may be appropriately considered in this place.

Ergot is a disease which affects many kinds of grasses, but it is especially common in rye: and the substance is hence called *Secale cornutum*, or ergot of rye. The diseased grain is developed into a kind of spur, growing in a curved form to the length of from half an inch to an inch or longer. The disease appears to be originally caused by a parsitical fungus (*Sphacelia segetum*,) [lately fully described by Mr. Quekett, (Trans. Lin. Soc. xviii.) as Ergotætia abortifaciens. See Griffith's Med. Bot., 676.—G.] and it is observed to prevail more particularly in damp seasons. The substance is of a deep reddish-black colour on the outside, and a reddish-white when fractured. It is brittle but tough, and, except when well dried, not easily pulverizable. It has a slightly acrid taste, and the odour when powdered, or brought out by potash, is nauseous and fishy. It forms a tincture of a dark-brown colour, which possesses a similar odour.

Its effects vary according to whether it be taken in large doses, or whether its use be long-continued in small doses. The results of numerous experiments on animals, and some observations on the human subject, show that ergot administered in a large dose, in any form (about two drachms of the powder,) is liable to occasion dryness and irritation of the throat, salivation, thirst, burning pain in the stomach, vomiting, colic, and sometimes diarrhœa. Cerebral symptoms, such as headach, giddiness, and stupor, are also met with. The appearances after death, have been in a few cases patches of inflammation on the mucous membrane of the stomach and small intestines.

The *chronic* effects of this poison have been witnessed occasionally on the continent in an epidemic form, and they have in some instances been distinctly traced to the admixture of ergot with rye-bread. In one set of cases the nervous system appears to be especially affected, indicated by vertigo, loss of sensation, tendency to sleep, rigidity of the muscular system, tremulous gait, and convulsions. After death the chief appearance consists of congestion in the brain, liver, and heart. In another set of cases the blood appears to undergo some change, hæmorrhages ensue;—black spots and boils appear in various parts of the body, and there is mortification of the extremities. After death the blood is found black and very fluid throughout the body. (Wibmer, Op. cit., *Sphacelia segetum*, 153.) These serious effects are not witnessed in this country, where rye-bread is but little used; and even on the continent, this condition, to which the name *ergotism* has been given, requires for its production, a very long-continued use of the diseased grain. M. Bonjean has given an account of two instances, in which spontaneous gangrene was brought on by bread containing ergot of rye. One child was ten years old, and it was found necessary to amputate both legs; the other, between two and three years old, lost the right leg by spontaneous separation. Other members of the family who had partaken of the food were indisposed, but no serious consequences ensued. The eldest child died in about a fortnight. On dissection the brain and its membranes were found healthy, but the meningeal veins were much distended. There was an effusion of serum in each ventricle. The other appearances were not remarkable. (Academy of Sciences, Dec. 1844; quoted by Dr. Beck, Amer. Journ. of Med. Science, July 1845, 243.) Mr. Nuttall has described a case which occurred recently in Ireland, in which it is probable that the singular symptoms observed in the patient, were owing to the effects of ergot. A man was admitted into Steeven's Hospital, Dublin, in June 1847. He complained of thirst, occasional cramps in the legs and feet, and loss of appetite. The nails had fallen off his thumbs and all the fingers of both hands, as well as one of the lesser toes of the left foot, the denuded parts being covered with flabby granulations secreting a fœtid discharge. In a few days these symptoms disappeared, but there was some redness about the parts surrounding the nails of both feet, with a slight reddish-purple discolouration. Suppuration gradually took place; the nails became thin, and one was removed. Under treatment, these, and the symptoms of nervous disorder, gradually ceased. On inquiry, it was ascertained that for six months he had been living much upon rye-meal of bad quality, grown in a poor marshy soil. About this period he had suffered from burning pain in his fingers, and suppuration took place; and three years previously, after eating the same kind of bread, his nails had fallen off, but with little pain or inconvenience. Some members of his family had suffered in like manner. (Dublin Med. Press, July 28, 1847, 53.)

The ergot, it is well known, is largely employed by accoucheurs to aid parturition, and indeed to bring it on. Much difference of opinion exists as to the abortive powers of this substance: some regard it as only acting on the uterus by the production of great constitutional injury, and that its effect is simply to accelerate, but not to induce labour. In many instances it has been found to bring on violent action of the uterus at an advanced stage of gestation, or when efforts at parturition had already commenced. According to Dr. Lee, it has no effect at least in the *early* stages of gestation, although given in very large doses. (Med. Gaz. xxv. 10; see also Ed. Med. and Surg. Journ. liii. 27.) Dr. Kluge, of Berlin, found that its properties varied according to whether it was gathered before or after harvest;—in the former case it had an energetic action, while in the latter it was powerless. The properties of the secale are not at all known to the vulgar; and this may account for the fact of our rarely hearing of cases where it has been criminally administered to pregnant females. Dr. Beatty has lately stated, that when used in obstetric practice it is liable, by absorption into the system of the mother, which may take place within two hours, to endanger the life of the child. (Dub. Med. Journ., May 1844, 202.) On trials for criminal abortion perpetrated or attempted, a medical witness must be prepared for a close examination on the specific emmenagogue properties of this and other drugs which may have been administered. A very instructive case occurred in 1844. (*Reg.* v. *Calder*, Exeter Lent Ass. 1844,) which has been ably reported with comments, by Dr. Shapter, Prov. Medical Journal, April 10, 1844. It was alleged in this case that savin, cantharides, and ergot had been respectively given by the prisoner, a medical man, for the purpose of procuring miscarriage. The prosecutrix was a woman of notoriously bad character, and the prisoner was acquitted. There were three medical witnesses, who agreed that savin and cantharides were only likely to occasion abortion, indirectly, *i. e.* by powerfully affecting the system—the view commonly entertained by professional men. Some difference of opinion existed with regard to *ergot*. Dr. Shapter stated in his evidence, in conformity with a general opinion, that he did not think the ergot would act, unless the natural action of the uterus had commenced,—a statement supported by a number of authorities. Subsequently to the trial, he collected the observations of many obstetric writers, and so far modified his opinion as to admit, that the ergot might *occasionally* exert a specific action on the uterus, in cases of advanced pregnancy, where uterine action had not already commenced. He has published a summary of opinions on this subject. (loc. cit.) Dr. Ramsbotham has reported three cases, from which it would appear that the ergot may in some instances exert a direct action on the impregnated uterus. In these instances, the females were in or about the *eighth* month of pregnancy. (Med. Gaz. xiv. 434.) Dr. J. H. Davis also believes that it is a specific excitant of uterine action, and points out the cases in which, in his opinion, it may be safely employed. (Lancet, Oct. 11, 1845, 393.) A large collection of cases, illustrating the properties of this drug, will be found in Wibmer. (Arzneimittel und Gifte. *Sphacelia segetum.*) The results of experiments on animals lead decidedly to the conclusion that ergot exerts a specific effect on the uterus, and the observations of Mr. Youatt fully corroborate this view. (Pereira, Mat. Med., ii. 919.) The conclusion appears to me to be, that, although in some instances ergot, even in large doses, may fail to excite uterine action, yet that in other cases it appears to act decidedly as an abortive, and to originate this action.

The medicinal dose of the powder as an emmenagogue is from five to fifteen grains, but its use should not be long continued. It is employed in a larger dose (twenty grains at intervals of half an hour) to excite uterine action. The dose of the tincture is a drachm; this is considered to be equal to twenty grains.

Ergot is not one of those poisons which easily causes death in one large dose: its fatal operation is more strikingly developed by its long-continued use in small doses. Its active properties are considered to be due to the presence of an oil, and not to an alkaloidal principle. This oil may be extracted by ether, and obtained by distillation.

ANALYSIS.—The external form is sufficient to identify ergot. In shape it is like a spur, somewhat curved, from half an inch to an inch long, having a black exterior, and breaking with a tough close fracture of a pinkish colour. Nitric acid gives to the powder a reddish tint, and a solution of caustic potash produces with it, even in the cold, a rich crimson-pink colour, becoming deeper when heated. It gives a nauseous smell to water and alcohol; but beyond this, I am not aware of any mode of pronouncing upon its presence in a state of solution. The tincture is of a dark-brown colour; potash does not act upon it as upon the powder, and there are no tests which can be safely applied for its detection under these circumstances. The powder, when heated, burns with a smoky yellow flame,, leaving a carbonaceous ash. (See Dunglison, New Remedies, 265.)

Other vegetable irritant poisons might be enumerated, but I believe these are the principal which have given rise, or are likely to give rise to medico-legal inquiries. The *treatment* of poisoning by the vegetable irritants, must depend on the nature of the symptoms; the main object, however, should always be to remove the substance from the stomach or bowels, with as little delay as possible. The nature of the poison is commonly apparent from the circumstances, these cases are generally the result of accident. These vegetable poisons are, as it has been already observed, in general beyond the reach of chemical processes:—they are only to be recognised either by their physical properties, or by the botanical characters of the berries, seeds, or leaves, with or without the aid of the microscope,

CHAPTER XXXIII.

ANIMAL IRRITANTS. CANTHARIDES, OR SPANISH FLY—SYMPTOMS—CHRONIC POISONING—EFFECTS OF EXTERNAL APPLICATION—POST-MORTEM APPEARANCES—QUANTITY REQUIRED TO DESTROY LIFE—TREATMENT—CHEMICAL ANALYSIS—PROPERTIES OF CANTHARIDINE—PHARMACEUTICAL PREPARATIONS—POISONOUS FOOD—POISONOUS FISH—MUSCLES—SYMPTOMS AND APPEARANCES—SUSPECTED DEATH FROM—TREATMENT—OTHER SHELL-FISH. POISONOUS CHEESE—SAUSAGES—POISONING BY THE FLESH OF ANIMALS—DISEASED MUTTON, VEAL, PORK, BACON—EFFECTS OF PESTIS BOVINA—OF PARTIAL DECAY—OF THE FOOD OF THE ANIMAL—POISONOUS MILK.

There are certain irritant substances belonging to the animal kingdom, which here require to be noticed, since they sometimes give rise to questions of poisoning. The first and most important of the animal irritants, is the blistering fly.

CANTHARIDES. SPANISH FLY.

Symptoms.—This poison has been frequently administered either in the state of powder or tincture, for the purpose of exciting aphrodisiac propensities, or of procuring abortion. When taken in the form of powder, and in the dose of one or two drachms, it gives rise to the following symptoms—a burning sensation in the throat, with great difficulty of swallowing; violent pain in the abdomen, with nausea and vomiting of bloody mucus; there is also great thirst, with dryness of the fauces, but, in a few cases observed by Mr. Maxwell, salivation was a prominent symptom. As the case proceeds, pain is commonly experienced in the loins, and there is incessant desire to void urine, but only a small quantity of blood or bloody urine is passed at each effort. M. Lavallée has lately observed, that one effect of the poison, externally applied, is to give a strongly albuminous character to the urine. (L'Union Médicale, 17 Juin, 1847, p. 308.) The abdominal pain becomes of the most violent griping kind. Diarrhœa supervenes, but this is not always observed:—the matters discharged from the bowels, are mixed with blood and mucus. In these as well as in the vomited liquids, shining green particles may be commonly seen on examination, whereby the nature of the poison taken, is at once indicated. After a time, there is often severe priapism, and the genital organs are swollen and inflamed both in the male and female. In one instance, observed by Dr. Pereira, abortion was induced, probably owing to the excitement of the uterus, from the severe affection of the bladder: for there is no proof that this substance acts directly on the uterus to induce abortion. With respect to the aphrodisiac propensities caused by cantharides,—these can seldom be excited in either sex, except when the substance is administered in a quantity which would seriously endanger life. When the case proves fatal, death is usually preceded by syncope, vertigo, and convulsions. The tincture of cantharides produces similar symptoms:—they are, however, more speedily induced, and the burning sensation and constriction of the throat and stomach are more strongly marked: these symptoms are often so severe as to render it impossible for the individual to swallow;

and the act of swallowing gives rise to the most excruciating pain in the throat and abdomen. The following well-marked case of poisoning by the *tincture*, occurred to Dr. Ives, of New York. A boy, aged seventeen, swallowed an ounce. When seen, an hour and a half afterwards, the respiration was hurried, there was profuse ptyalism, convulsive trembling, acute pain in the region of the stomach and bladder, and such exquisite sensibility that the slightest pressure produced convulsions. These came in paroxysms, were accompanied by painful priapism, and followed by delirum. On the seventh day, he was seized with pain in the head, trembling and universal spasms; coma followed. He then appeared to improve, but on the fourteenth day violent convulsions supervened, and these were followed by insensibility and death. (Beck's Med. Jur. 5th Ed. 842.)

Chronic Poisoning—It is not often that we have occasion to observe poisoning by cantharides in a chronic form, but a remarkable set of cases has been reported by Mr. Frestal, which show that, contrary to common belief, the substance does not invariably excite those aphrodisiac propensities which have been generally ascribed to it. It appears that six young men (students) had during a period of six months, unknowingly taken with their food, powdered cantharides by mistake for pepper. The quantity taken was at no time large, but very variable. The only marked general symptom was, great restlessness. There was no affection of the nervous system, nor any disorder of the bowels. The appetite was unaffected, No pain was experienced in the renal or lumbar regions. About three hours after the meal, there was a slight pruritus of the glands, with a desire to micturate, and there was also *ardor urinæ*. The desire for micturition continued for from two to four hours, and then gradually ceased, leaving some irritation about the urethra. There was neither priapism nor any erotic feeling. The absence of the symptoms is the more remarkable, as the substance must have been taken in very different doses at different times. Without knowing the cause of the disorder from which they suffered, they employed for their relief, warm baths, and an abundance of warm demulcent drinks. This plan of treatment was found to be most effectual. (Journal de Chimie Médicale, Janvier 1847, p. 17.)

Effects of external application.—It is proper to state that cantharides will operate as a poison, when applied externally to a wound or ulcerated surface, or even when applied to a large surface of healthy skin. In January, 1841, a girl, aged sixteen, was killed at Windsor, under the following circumstances, She was affected with the itch,—sulphur ointment was prescribed for her; but by mistake, blistering ointment was used. This was rubbed all over the body of the girl:—she was soon seized with the most violent burning pain,—the ointment was immediately washed off, but the cuticle came off with it. The girl died in five days, having suffered from all the usual symptoms of poisoning by cantharides.

POST-MORTEM APPEARANCES.—In one well-marked case of poisoning by this substance, the whole of the alimentary canal from the mouth downwards, was in a state of inflammation, as well as the ureters, kidneys, and internal organs of generation. The mouth and tongue seemed to be deprived of their mucous membrane. In Dr. Ives's case, above mentioned, where an ounce of the tincture was swallowed, and death did not occur for fourteen days, the mucous membrane of the stomach was not inflamed; but it was pulpy and easily detached. The kidneys were, however, inflamed. The brain has been found congested, and ulceration of the bladder is said to have been met with. There are very few fatal cases reported, in which the appearances have been accurately noted; indeed, the greater number of those who have taken this poison, have recovered. Cantharides are sometimes described as a corrosive poison; but the substance appears to have no local action of a chemical nature. It is a pure *irritant*, and the effects observed are entirely due to irritation and inflammation. Serious accidents have frequently occurred from the powder of cantharides being mistaken for jalap, cubebs, and other medicinal substances. A man lost his life lately by having cantharides-powder supplied to him for cubebs, in a druggist's shop.

QUANTITY REQUIRED TO DESTROY LIFE.—This has been a frequent subject of medico-legal inquiry. Dr. Thomson represents the medicinal dose of the powder to be from one to three grains. On a late criminal investigation, the medical witness stated, that one grain was the maximum dose, but this is an under statement; according to Thomson it is *three* grains:—the dose of the London Pharmacopœial tincture is from ten minims gradually increased to one fluid drachm,—of the powder, from *one to two grains*. (Pereira, Mat. Med. ii. 1846.) Doses above this, whether of the powder or the tincture, are likely to be injurious, and to give rise to symptoms of poisoning. On a trial which took place at Aberdeen, in 1825, it appeared that a drachm of the powder had been administered: severe symptoms followed, but the person recovered. Dr. Dyce, the medical witness, said he had given ten grains of the powder at a dose as a medicine. In three cases, observed by Mr. Maxwell, a drachm of the powder mixed with six ounces of rum was taken by each person: they were robust, healthy negroes,—they suffered severely, but recovered in about ten days:—in these cases, irritation of the urinary organs did not appear until after the men had been bled. The *smallest quantity* of powder which has been known to destroy life, was in the case of a young female, quoted by Orfila,—the quantity taken was estimated at *twenty-four grains* in two doses. She died in four days; but as abortion preceded death, it is difficult to say how far this may have been concerned in accelerating that event. Her intellect was clear until the last. In one instance a man recovered after having taken twenty grains of the powder, (Ed. Med. and Surg. Jour. October, 1844;) but in another still more remarkable case, which occurred to Dr. Fisher, a man, aged twenty-six, recovered after having taken upwards of sixty grains. The powder was swallowed by mistake for jalap. Some hours afterwards, the patient was found labouring under incessant vomiting, intense thirst, with burning pain in the mouth, throat, and stomach, countenance anxious, tongue swollen and thickly coated, pulse 130, weak and tremulous:—the matter vomited had a greenish colour, and a peculiarly offensive odour. There were frequent and urgent calls to micturition, always preceded by severe pain at the point of the penis; and the passage of the urine was attended with severe scalding pain. The urine was turbid, and slightly tinged with blood. There was a dull heavy pain in the lumbar region, increased by pressure: and occasional priapism. Vomiting was promoted, and a large quantity of thick solution of gum-arabic was administered at intervals. The patient rapidly recovered, (Med. Gaz. xxxix. 855,) and his recovery was probably due to the greater part of the poison having been ejected by the early occurrence of vomiting.

An *ounce* of the tincture has been known to destroy life in fourteen days. This I believe is the *smallest dose* of the *tincture* which has killed. (Dr. Ives's case, p. 542.) In the following instance a similar dose produced only serious symptoms. A woman, aged twenty-nine, swallowed an ounce of tincture of cantharides. Some time afterwards, there was severe pain in the abdomen, increased by pressure: it became swollen and tympanitic. She passed during the night a pint and a half of urine unmixed with blood. In two days, the pulse became feeble and scarcely perceptible:—there was delirium, with severe pain in the region of the kidneys and bladder:—the urine was continually drawn off by a catheter. It was more than a fortnight before she was convalescent. (Med. Gaz. xxix. 63.) Four drachms and even six drachms have been taken; and although the usual symptoms followed, the parties did well. A case of poisoning by cantharides was the subject of a trial at the Central Criminal Court, in September, 1836. Six drachms of the tincture were administered to a girl, aged seventeen: the medical witness was required to say whether half an ounce was sufficient to kill, as also what proportion of cantharides was contained in the tincture.

One ounce of the tincture, P. L., is equivalent to six grains of the powder; but considering that the principle *Cantharidine* is the substance on which the poisonous properties depend, it is very likely that the tincture may vary in strength according to its mode of preparation. A case is quoted by Pereira, from Dr. Hosack, (Mat. Med. ii. 1842, in which it is stated, that six ounces of the tincture were taken by a man without causing dangerous symptoms! This must have been an extraordinarily weak preparation: and probably the insects from which the tincture was made, contained little or no cantharidine. The same writer mentions a case within his own knowledge, in which one ounce of the tincture caused serious symptoms.

[The case quoted by Pereira is related by Dr. Hosack, in the appendix to Thomas's Practice, 1036, and there is nothing but the impunity with which so large a dose was taken, to lead to a belief that the tincture was inert. From our own experience, we are persuaded that no rule can be laid down as to the quantity that may prove injurious; some persons appear to be but little affected by large doses, whilst others are extremely susceptible to its action.—G.]

Cantharides are sometimes taken in the form of blistering plaster. A case was reported to the Westminster Medical Society, in which a woman took a piece about the size of a walnut, in chocolate, by mistake. In about an hour, vomiting and strangury supervened: this was followed by inflammation of the kidney. The woman speedily recovered. In another instance, in which half an ounce of the plaster, containing two drachms of the powder, was taken, death took place in twenty-four hours. (Ed. Med. and Sur. Jour. October 1844.) A singular case, in which an attempt was made to poison a man by blistering plaster, was recently the subject of a trial in France. This person perceived after taking some soup, a strong and bitter taste, for which he could not account. He also suffered from violent pain in the stomach and abdomen, especially in the region of the bladder; and he could only under scalding pain, void a small quantity of urine, tinged with blood. He recovered from these symptoms, but three months subsequently, and two hours after taking some soup which had the same bitter taste, they returned in an aggravated form. They were relieved by doses of olive oil and milk to excite vomiting; a few days afterwards, he found in this soup a dark-coloured substance, which on examination by a medical man, turned out to be cantharides. His brother-in-law, who was proved to have recently purchased blistering plaster, was tried on a charge of attempting to poison. One half of the plaster sold to the prisoner, was found, and it was proved to contain about thirty-one grains of powdered cantharides. The medical witnesses agreed that the symptoms under which the prosecutor had laboured, were those which commonly result from this poison; but one of them contended that the dose administered was not sufficient to cause death! (The exact quantity taken is not stated.) The accused was nevertheless capitally convicted. (Journal de Chimie, 1846, p. 606.) In the Registration returns for 1840, one case of death from cantharides is recorded, in a male aged 46.

TREATMENT.—When vomiting exists, this may be promoted by warm demulcent liquids, as thick linseed-tea, or a very strong solution of gum-arabic: if it does not exist, emetics should be given,—the object being to dislodge the poison. Demulcent enemata may also be used. The state of the throat will scarcely admit of the application of the stomach-pump. Oil was formerly regarded as an antidote; but it has been found that this is a ready solvent of the active principle, and it is therefore injurious.

[It has been stated by Devergie and others that camphor, although not an antidote to this poison, acts efficaciously in alleviating some of the most unpleasant symptoms.—G.]

CHEMICAL ANALYSIS.—*Cantharidine* is the vesicating, and at the same time the poisonous principle of the insect. It is a white solid crystallizable substance, insoluble in water; but soluble in ether, alcohol, fixed oils and caustic alkalies. Although water does not dissolve it in its pure state, it takes it up with other principles, from the powdered insect; and thus an infusion of cantharides is poisonous. It is very volatile, and produces serious effects in the state of vapour. There are no chemical characters by which this principle can be safely identified, if we except its *vesicating* properties. Orfila has applied reagents to detect cantharidine in the tincture; but without success. It has been recommended to digest the suspected solid, or the liquid contents of the stomach evaporated to an extract, in successive quantities of ether,—to concentrate these ethereal solutions by slow evaporation, and then observe, whether the concentrated liquid produces vesication or not:—the medical jurist being expected, in such cases, to make himself the subject of experiment. In this way, Barruel discovered cantharides in some chocolate. (Ann. d'Hyg. 1835, 455.) This mode of testing is somewhat uncertain, unless the quantity of poison be large; and the affirmative evidence which it yields is better than the negative; since we can hardly infer the absence of the poison, when we obtain no result. There is, however, no other mode of discovering cantharides in solution, whether as tincture or infusion, than this.

The difficulty of extracting *Cantharidine* may be conceived, when it is stated that, according to Thierry's experiments, which are the most perfect, the quantity contained in the fly is only about the 250th part of its weight, so that it would require nearly half an ounce of the powder, to yield one grain of cantharidine. The quantity required to produce vesication is unknown, but it is extremely small. Cantharides are most commonly taken in powder, and then we may easily recognise the poison by its physical characters. If the insect be entire, or only coarsely powdered, there can be no doubt of its nature. However finely reduced, the powder is observed to present by reflected light, small golden green or copper-coloured scales. These are perceptible to the eye, and are very distinct under a common lens. It has been recommended to separate the particles of cantharides, by suspending the liquid or other contents of the stomach in warm water, when the insoluble powder will subside, and the particles may be collected and dried for examination. In an elaborate essay on this subject (Ann. d'Hyg. Oct. 1842,) M. Poumet recommends that the suspected liquids, mixed with alcohol, should be spread on sheets of glass, and allowed to evaporate spontaneously to dryness. The shining scales will then be seen, on examining by reflected light either one or both surfaces of the glass. This experiment answers very well. He has also found that the particles adhering to the mucous membrane of the stomach or intestines, may be easily detected by inflating the viscus, and allowing it to become dry in the distended state, taking care to attach to it a heavy weight, so that during the process of drying, all the folds of the mucous membrane may disappear. On cutting the dried membrane and opening it on a flat surface, the shining scales become perceptible. Physical evidence of this kind would not be of much avail for medico-legal purposes, unless there were concomitant evidence from symptoms and post-mortem appearances. In trials for administering this poison, the analysis might be confined to the article administered; and the physical test is then applicable, since the powder is commonly given in very large quantity, and adheres closely to the mucous membrane. There are many insects, besides cantharides, which have wings of a golden-green colour, and are not poisonous: yet such insects are not likely to be found in the form of powder in the human stomach. M. Poumet states that there are some cantharides which contain no cantharidine.

The evidence of the presence of cantharides, or of their having been taken, is necessary to support a criminal charge; for, however unambiguous the symptoms produced by this poison, may appear to be in its peculiar effects on the generative and urinary apparatus, the medical jurist should be aware that similar symptoms may proceed from disease. An important case of this kind has been published by Dr. Hastings. (Med. Gaz. xii. 431.) A young lady was suddenly seized with vomiting, thirst, pain in the loins, strangury, and considerable discharge of blood from the urethra: the generative organs were swollen and painful. She died in four days. She was governess in a family, and there was some suspicion that she had been poisoned by cantharides. The stomach and the kidneys were found inflamed, and the bladder also: this organ contained about two ounces of blood. There was no trace of poison, and indeed it was pretty certain, from the general evidence, that none could have been taken.

Particles of cantharides may be detected in the viscera long after interment. Orfila has detected them after a period of nine months, so that they do not seem to be affected by the decomposition of the body.

PHARMACEUTICAL PREPARATIONS.—The *doses* and *comparative strength* of the powder and tincture of Cantharides according to the London Pharmacopœia, have been already stated (p. 438.) There are some other preparations, the strength of which may be important for the medical jurist to know. The ACETUM CANTHARIDIS or Vinegar of Cantharides is used externally. It is equivalent to about one-tenth of the powder; *i. e.* five ounces are equal to four drachms of powdered cantharides. The CERATUM CANTHARIDIS contains one-sixth, and the EMPLASTRUM CANTHARIDIS contains one-half of its weight of the powder.

[There are several species of blistering flies, found in the United States, possessing all the properties of cantharides. See Journ. Phil. Coll. Pharm., I., 276.—G.]

POISONOUS FOOD.

Certain kinds of animal food are found to produce occasionally symptoms resembling those of irritant poisoning. In some instances, this poisonous effect appears to be due to idiosyncrasy; for only one person out of several may be affected. These cases are of some importance to a medical jurist, since they are very likely to give rise to unfounded accusations of criminal poisoning. In the absence of any demonstrable poison, we must test the question of idiosyncrasy by observing whether more than one person is affected, and whether the same kind of food, given to animals, produces symptoms of poisoning: if, with this latter condition, several persons be affected simultaneously, we cannot refer the effects to idiosyncrasy; they are most probably due to the presence of an animal poison. There may, it is true, be a family idiosyncrasy, but this is not very common. Among the articles of food which have given rise to symptoms of poisoning, we may first mention

POISONOUS FISH.

Of all the varieties of shell-fish, none have so frequently caused accidents as the COMMON MUSCLE (MYTILUS EDULIS.)

SYMPTOMS AND APPEARANCES.—The symptoms which it produces are swelling of the face, uneasiness and sense of weight in the epigastrium, sensation of numbness and tingling in the extremities, heat, and constriction in the mouth and throat; thirst, rigors, difficulty of breathing, cramps in the legs, swelling and inflammation of the eye-lids, with a profuse secretion of tears, and heat and itching of the skin, followed by an eruption resembling urticaria. The symptoms are sometimes accompanied by burning pain in the abdomen, vomiting, colic, and diarrhœa. They may occur within ten minutes or a quarter of an hour; but their appearance has been protracted for twenty-four hours. There is in general great debility. These effects have proceeded from the eating of not more than ten or twelve muscles. Two cases, which occurred to Dr. Combe, proved fatal, the one in three, and the other in about seven hours (E. M. and S. J. xxix. p. 86;) and two others have been reported by Dr. Burrows. In general, however, especially where there is free vomiting, the patients recover. In the inspection of Dr. Combe's two fatal cases, no appearance was found to account for death. In a recent case which occurred to Dr. Barclay, death was obviously due to peritonitis. A man, æt. 24, procured half a gallon of muscles, and ate about two-thirds, previously roasted, in the evening. It was not until the following morning that he began to experience symptoms of uneasiness, and he soon felt the most excruciating pain in the abdomen. Purgatives were given, but no active treatment was adopted for three days, when he was found labouring under symptoms of peritonitis. He died about a week after he had eaten the fish; and, on inspection, the whole of the peritoneal surface was injected, and adherent to those parts of the intestines which were in contact with it,—lymph and pus being effused in several places, but more particularly about the folds of the jejunum. The stomach was empty, more vascular than natural, but not inflamed. The mucous membrane of the duodenum was much injected, being filled with a glairy dark thick matter, like inspissated mucus. The mucous membrane of the jejunum was also injected; the rest of the body healthy. A companion who had partaken of the muscles also suffered, but in a less degree. (Prov. Med. Journ., Dec. 10, 1845, 722.) From the enormous quantity of muscles eaten in the above case, it might be a question whether the indigestible animal matter had not acted by mechanical irritation. The peculiar symptoms indicative of an affection of the brain and nervous system, as well as those arising from irritation of the skin, were entirely wanting.

It is not often that a medico-legal question can arise in reference to this form of poisoning; but the following suspicious case occurred not long since to my friend Mr. French, who communicated to me the particulars. M. S., aged 33, had eaten for breakfast, at nine o'cock, abour four muscles, with bread and butter, and had taken part of a pint of beer. About eleven she went out, but after walking a short distance she was seized with giddiness, and fell. She walked home with assistance, after which she recovered her senses sufficiently to say to a bystander, "I am dying." She had been in this state of illness for about an hour, when Mr. French was called to her, and he found her dead; but she had died so calmly that her death was not even known to have taken place by those who were around her. He ascertained that there had been violent vomiting, and that the deceased had complained of headach and pain in the stomach. On an examination of the body, the only appearance was a highly congested state of the vessels of the brain and its membranes. There were firm adhesions of the pleura on the left side of the chest. The right side of the heart was rather thinner than normal; the liver was somewhat hypertrophied. The stomach contained alimentary matter nearly digested. It seems doubtful whether death could be attributed to the muscles in this instance, from the quantity eaten being so small; and yet no other cause was apparent.

The following well-marked case of poisoning by muscles (*Mytilus edulis*,) was reported in the Lancet by Mr. W. Cooper (March 7, 1846, p. 274.) A man aged 20, ate about fifty muscles. In about half an hour he was seized with alarming symptoms. His face was deeply flushed, almost purple, eyes staring and intensely congested, and the pupils dilated. His breathing was stertorous, and was performed with great effort; pulse slow and labouring, and he seemed partially comatose. A mustard emetic was given, which emptied the stomach. In about twenty minutes he began to complain of intense itching, and the whole of the upper part of the body was found to be covered with an urticarious eruption, of a deep crimson hue. His feet were cold, and he was unable to stand, having lost all power in his legs. He complained of numbness in his face and throat, with an intense pricking sensation about the forehead and temples. There was intense thirst with great debility. These symptoms did not abate for three days, and he then began slowly to recover. He stated that while the attack was on him, he was for three hours perfectly blind, and seemed to have lost all his faculties. He had neither the power of thought nor of memory. The interior of his mouth, head, and throat, seemed swelled to twice their natural size, and his eyes felt as if they were starting out of their sockets. In breathing, it appeared to him as if he were drawing air through a sponge. He had no particular uneasiness in the abdomen at any time, nor was there strangury or irritation about the urinary organs. Three other persons who partook freely of the muscles merely suffered from indigestion: they experienced no symptoms of poisoning.

[Many cases of an analogous character have occurred in New York, and Dr. Lee in his notes to Guy, *Princ. For. Med.* 650, states, he knew of a death in a family of seven, who were all poisoned by these shell-fish.—G.]

TREATMENT.—The treatment of poisoning by muscles consists in the free exhibition of emetics. The poisonous action of muscles can neither be referred to putrefaction nor disease; nor in all cases to idiosyncrasy, since sometimes those muscles only have been poisonous which were taken from a particular spot; all persons who partook of them suffered, and a dog was killed to which some of them were given. From a case which occurred to M. Bouchardat, it would appear that copper is sometimes present, and may be the cause of the poisonous effects. Two women were poisoned by muscles, and he found on analysis sufficient copper in the fish to account for the symptoms of irritation from which they suffered. (Ann. d'Hyg. 1837, 358.) Copper is not however, present in all cases, and it is therefore probable that there is in some, if not in all instances, an *animal poison* present in the fish. WHELKS, which are largely sold in the streets of London, may produce effects similar to those caused by muscles.

OYSTERS and PERIWINKLES have occasionally given rise to similar symptoms. SALMON, sold in the state of pickled salmon, or even HERRINGS salted, may also act as irritants; this may be due to the fish being partially decayed before it is used. Herrings have been known to produce violent symptoms of irritation, but this has been commonly traced to the liquid used for pickling them. M. Fayrer met with the following case. A woman, aged 30, ate for her dinner some herrings, which, although well washed and cleaned, exhaled a nauseous odour, and had a very disagreeable taste. She was soon afterwards seized with symptoms of poisoning; there was a feeling of oppression at the epigastrium, intense thirst, with a sense of coldness and depression. These symptoms were followed by vertigo, so that she could not stand, dilatation of the pupils, imperceptible pulse, cold perspiration, and loss of consciousness. While being carried into the open air, she vomited a colourless mucous liquid, and the symptoms then abated. After this, at intervals of a few seconds, there came on the most violent paroxysms of pain in the abdomen; the features were contracted, and the lips and teeth spasmodically closed. There was nausea, but no vomiting; the pulse weak and thready, and the whole of the surface still bathed in a cold perspiration. The woman refused to drink. In about half an hour the symptoms subsided, and she had a calm sleep. She recovered the day following. Two other persons, who had eaten a small portion of the same herrings, merely experienced for several hours, oppression of the stomach. (Journ. de Chimie, 1845, 654.) In 1834, two persons at Maidstone lost their lives from eating pickled salmon.

POISONOUS LOBSTERS.—The following case of alleged poisoning by lobsters is reported in the Medical Gazette (vol. ii. 320.) The effects were most probably due to mineral poison. A cargo of lobsters was lately brought to Carlsham, and seventy-four persons who partook of them were seized with sickness and other symptoms resembling those of mineral poisoning. The Russian Vice-consul and two other persons died. Search was made for the owner of the vessel who had brought them, but he had left and could not be found. A ship-captain, who was brought up to the lobster fishery, and who resided at Carlsham, stated that when lobsters were sent to a distant part, it was usual to parboil them, and to strew each layer with salt and a little mercury (arsenic?) to keep them fresh! A strict investigation was ordered, but the result did not transpire. Several persons were taken ill, and one died in a few hours at Christianstadt, where the master of the vessel sold the first part of his cargo.

LOBSTERS, CRABS, and CRAYFISH have been known to produce severe irritation, sickness, and purging in cases where no suspicion of the presence of mineral poison could be entertained.

The liver of the HALIBUT, caught off New York, in one instance related by Dr. Beck, caused pain, nausea, vomiting, and headach, followed by exfoliation of the skin. (Med. Jur. 853.)

[Another case from eating Halibut is reported in New York Med. Gaz. II.—G.]

The edible fish, caught off our coast, cannot be regarded as poisonous. In tropical seas there are, however, numerous species which are capable of producing severe symptoms and speedy death. These it is unnecessary to enumerate. The cause of this poisonous property has been assigned to the food taken by the fish (acrid mollusca,) but this is a mere hypothesis.

The *symptoms* caused by fish-poison resemble those described in speaking of the common muscle. In addition to symptoms of irritation, giddiness, unconsciousness, coma, and convulsions often precede death. The following is a recent case of poisoning by TUNNY (*Tetrodon Sceleratus*,) reported by Dr. Galiay of Tarbes. Eight persons sat down to dinner in perfect health. Among the dishes was a tunny—a large species of *mackerel*—found in the Mediterranean. Several of those present remarked that it had an unusual taste. All suffered from symptoms of poisoning in different degrees, with the exception of two who took coffee immediately after the meal. The first and most general symptom was immediate irritation of the mouth, accompanied by an eruption of small vesicles in some, and by swelling of the gums and lips in others. The vesicular eruption soon passed away. The face, eyes, and ears, then became red, almost of a deep purple colour: this symptom was followed by intense headach, vertigo, and noise in the ears. The headach, which continued for some time, had remissions. An eruption, resembling urticaria, appeared on the skin of the neck and chest, extending afterwards to the other parts of the body: this was accompanied by intense itching. A dog and a cat, which had eaten part of the fish, suffered from severe vomiting and purging. In the absence of medical assistance, various popular remedies were resorted to, such as exposure to fresh air, cold affusion, emetics, and a mixture of vinegar and water. This is said to have given great relief. Of five other persons who partook of the fish, similar symptoms occurred in three; the two who escaped had taken coffee. A cat which ate up the residue of the meal, suffered severely from its effects. Many similar cases of poisoning by this fish occurred in the neighbourhood. Equally noxious effects have been observed to result from the use of the fish, whether it was in a perfectly fresh state or had been kept some days. Dr. G. observes, that on all these occasions, coffee was found to be an excellent restorative. (Gaz. des Hôp. Mars. 1846.)

The fish here referred to is known to the French under the name of "Thon" (tunny,) and abounds in the Mediterranean; it is allied to the mackerel genus, but is not known as an edible fish in England. The tunny is said to be met with on the western coasts of Scotland, and is called mackerelsture, or great mackerel. This fish is not described by toxicologists as poisonous. From the uniformity of attack, and the number of persons who suffered on this occasion, it is evident that the symtoms could not have been due to idiosyncrasy, but to the presence of some animal poison. The fact also that animals which partook of the fish equally suffered, proves that it must be possessed of directly poisonous properties. The symptoms were very analogous to those produced by other kinds of fish-poison. The coffee probably acted as a stimulant, and not as an antidote. In treating these cases, we can only trust to active emetics and purgatives, or enemata.

According to Dr. Beck, the MACKEREL of New York has occasionally produced poisonous effects. (Med. Jur. 853.) This fish is very largely consumed in England: but so far as I know, no dangerous symptoms have ever resulted from its use. EELS sometimes give rise to nausea, pain, and vomiting, effects probably due to idiosyncrasy. The CONGER-EEL of the tropics (MURÆNA MAJOR) is decidedly poisonous: but the YELLOW-BILLED SPRAT) CLUPEA THRYSSA) appears to be the most fatal to human life. This fish is confined to the tropics.

CHEESE. SAUSAGES.

These articles of food have frequently given rise to symptoms of poisoning in Germany, but there is, I believe, no instance of their having proved fatal in England. The symptoms produced by cheese have been those of irritant poisoning. The nature of the poison is unknown. In examining several specimens of decayed cheese, I have found in them only an acrid oil and sesquicarbonate of ammonia. In some cases the poisonous property is undoubtedly due to a putrefied state of the curd. Again, it has been supposed that the poison is occasionally derived from certain vegetables on which the cows feed. The symptoms caused by the *Sausage-poison* are very slow in appearing; sometimes two, three, or four days elapse before they manifest themselves—they partake of the narcotico-irritant character. This poison is of a very formidable kind. In the Medical Gazette for Nov. 1842 (xxxi. 272,) there is an account of the cases of three persons who had died from the effects of liver-sausages, which had been made from an apparently healthy pig, slaughtered only a week before. The inspection threw no light on the cause of death. The poisonous effect is supposed to depend on a *partial* decomposition of the fatty parts of the sausages. It is said that when extremely putrefied, they possess no poisonous properties!

TRAIN OIL.

I am not aware that this oil has acted as an irritant on man, but it has caused the death of cattle within a quarter of an hour, producing intense suffering and foaming at the mouth. After death the intestines were found inflamed. The oil was not pure train oil, but a mixture of naphtha and fish oil. (Pharm. Journal, April 1845.)

POISONING BY THE FLESH OF ANIMALS.

Diseased Mutton.—The following case, which was referred to me for examination in December 1840, will illustrate the effects sometimes produced by ordinary articles of animal food:—

Three members of the family of a shepherd—the wife, son, and daughter, the two latter being young children—were taken ill on Sunday, December 20th. The boy, who was about two years old, died the following day. It was supposed that mineral poison had been administered to the family, and that this was the cause of the boy's death. The poison was suspected to have been taken at dinner, about 11 A. M. on Monday, December 21st, when all three dined with the father, on some mutton. No satisfactory history could be procured of the symptoms suffered by the wife and two children on the Sunday,—the day preceding. The only account that could be obtained was, that the body of the deceased child was swollen all over. The three were, however, better on the Monday. Having dined at the hour above mentioned, and the father having left for his usual work, they were not seen until about two o'clock, when the mother and daughter were in a state of insensibility, and the boy was dead. The following account was obtained:—The father stated, that after he had dined with the family on the Monday at eleven o'clock, he felt, while at work, a sharp burning pain in his inside, for which he could not account. This was between the time of his leaving dinner and two o'clock in the afternoon. The mother, on her recovery, said that she felt great pain after the meal; but no other account of her symptoms could be procured, except that she foamed at the mouth, and was in a state of great nervous excitement. So far as could be ascertained, she had suffered but slightly from vomiting, and there was no purging. The deceased and his sister were, however, affected with vomiting and purging. The deceased child died in *less than three hours* after the meal, for he was found quite dead at two o'clock; but no satisfactory account of his symptoms before death could be procured. It appears, however, that he was very much purged, and that his motions were of a dark-green colour. The matters vomited by him were very copious, and streaked with a yellow-coloured substance: these were, unfortunately, thrown away. The matter vomited by the mother is described as having had a bright glistening appearance, like quicksilver, on the surface. The stomach-pump was applied to the mother about six hours after she was found (eight o'clock P. M.;) and the contents of the stomach then drawn off were reserved for a chemical examination.

The following appearances were met with, on a post-mortem examination of the body of the deceased child. There was no particular appearance externally, except that the cutaneous surface was unusually pallid. The lungs were loaded with blood of a scarlet colour. The heart was natural: the liver of a pink colour, congested with very liquid blood. The stomach contained a small quantity of half-digested food, presenting, on its posterior part, several prominent rugæ, which were inflamed, with traces of inflammation on other parts of the lining membrane. The small intestines were inflamed in their upper portion; but the appearance of inflammation was less marked inferiorly. They contained a liquid mixed with blood. The muscular coat of the rectum was very red; but there was nothing in other respects abnormal, either in the large intestines or their contents. The peritoneum was highly inflamed. The bladder was contracted; and on its posterior wall were two spots of well-defined inflammation. The spleen and kidneys were healthy. About two ounces of bloody serum were found in the cavity of the abdomen. The upper part of the larynx and lower part of the pharynx were inflamed; and there were traces of inflammation at the bifurcation of the trachea. The veins of the head were very full, as well as those on the surface of the brain. The brain was of large size, and well developed: its vessels were congested; but there was no morbid change of structure. The contents of the stomach and viscera were collected, and forwarded to a chemist at Cheltenham for analysis. No trace of poison could be detected in them.

The suspicion of poisoning appears to have arisen from the sudden and violent illness of three persons after a meal, and the rapid death of one; as well as from the fact that the man and his wife lived somewhat unhappily together, the woman having had an illegitimate child (the daughter, who also suffered) by another man. No account was furnished of the symptoms under which the daughter laboured; except that, like the deceased child, she was affected with vomiting and purging. It appeared that the mutton that the family had had for dinner on the Monday, was part of the body of a sheep which had been affected with "the staggers," and which, in consequence, had been killed, and the meat distributed among many poor families in the neighbourhood. It was therefore not unreasonably considered that the very unwholesome nature of this food might sufficiently account for the serious consequences which had followed the meal. It is, however, worthy of remark, that no other persons of other families, who had freely partaken of the mutton from the same sheep, were attacked, or experienced any ill consequences whatever; this was the only family which suffered. Hence it follows that the effects were due either to idiosyncrasy or to the decayed state of the food. A strict investigation of all the facts, rendered the latter view the more probable. (G H. R. April 1843, p. 1.)

Flesh of over-driven animals.—It is said that the flesh of animals over-driven, as well as newly-killed meat in general, is liable to produce violent gastric irritation, and even cholera. (See ante, p. 50) What the effect of particular epizootic diseases may be, it is not easy to determine, seeing that there is a great want of observations on this point; but as a general principle, it appears to me that we shall be justified in admitting that the flesh, as in the pestis bovina, must more or less partake of this diseased state of the animal, and thus be unfitted to serve as an article of human food. Flesh of this kind is liable to become putrefied sooner than that of healthy animals; and its poisonous properties may, in a certain degree, be aggravated or brought out by this condition, With regard to putrefaction, it would appear that the flesh of the most healthy animal is rendered unwholesome by this process; but that the most severe effects are produced by that flesh which has become only *partially* decomposed: where, in short, putrefactive fermentation has been recently set up. Cases of poisoning from diseased or putrefied mutton or beef are by no means common. Veal, pork, and bacon, have, however, frequently given rise to alarming symptoms of gastric irritation and death.

Veal.—Dr. Christison relates the following case of poisoning by veal:—In the autumn of 1826, four adults and ten children ate at dinner a stew made with meat taken from a dead calf which was found by one of them on the sea-shore, and of which no history could be procured. For three hours no ill effects followed. But they were then all seized with pain in the stomach, efforts to vomit, purging and lividity of the face, succeeded by a soporose state, like the stupor caused by opium, except that when roused the patients had a peculiarly wild expression. One patient died comatose in the course of six hours. The rest having been freely purged and made to vomit, eventually got well; but for some days they required the most powerful stimulants, to counteract the exhaustion and collapse which followed the sopor. The meat, they said, looked well enough at the time it was used; yet the remains of the dish which formed the noxious meal had a black colour and a nauseous smell; and some of the flesh which had not been cooked had a white glistening appearance, and was so far decayed that its odour excited vomiting and fainting. (On Poisons, 647.) Dr. Christison thinks that the body of the animal in this case had lain for some time in contact with water, and that it had begun to undergo the process of saponification. The adipocere produced may have fermented; and this may have led to the formation of the same poisonous principle as that which is produced in the fermentation of German sausages. The only certain fact, however, upon which we can rely, is, that this veal acted like a narcotico-irritant poison, either from the flesh of the animal being previously diseased, from incipient decomposition, or from both causes conjoined. This gentleman also relates a case which occurred in Macclesfield, in 1830, in which the symptoms of poisoning were traced to some unsound *beef*.

Although this poisonous action of animal food has been observed to be connected more with the fatty than the lean parts of the flesh, yet it is by no means necessary for this action to follow, either that fat should be present, or that it should have begun to take on the adipocerous transformation. The following case, which is of some interest, was published in the Journal de Pharmacie et de Chimie, Aug. 1842:—At a public festival at Zurich, in the year 1839, upwards of 600 persons partook together of a repast, consisting chiefly of veal, roasted or in cutlets. At *variable periods* afterwards, nearly all of these individuals were taken ill; and in a week most of them were confined to their beds. They were affected with rigors, vertigo, headach, intense fever, diarrhœa, vomiting, and in some instances, delirium. At a later period an abundant flow of fœtid saliva occurred, the interior of the mouth being covered with ulcers; and in many cases, after involuntary discharges of the fæces,—great prostration of strength and death, ensued. In these cases, the mucous membrane of the digestive canal was found softened, the intestinal follicles ulcerated, and the veins empty. It was afterwards ascertained that the veal, when eaten, had been in an *incipient* state of putrefaction. The great analogy between the symptoms produced by it, and those of typhus, was very striking; and it is further remarkable, that many of the persons who attended on the patients, though previously healthy, and who had not been partakers of the deteriorated food, were attacked by the disease. In this case the poisonous action of the veal could only be attributed to incipient putrefaction. The symptoms were those of narcotico-irritant poisoning; and the exact period of the attack after the meal, appears to have been different in the different cases.

In the same journal it is related that the following circumstance occurred recently in the Grand Duchy of Baden:—A roebuck, having been taken in a net, was killed while making violent efforts to escape, and while in a state of the utmost terror and exhaustion. Nearly all the persons who partook of the flesh of this animal experienced a violent gastro-intestinal inflammation, with other symptoms similar to those detailed above; although, in this instance, the flesh was neither in a putrefying state, nor were any of the cases fatal. This last case confirms the general opinion, that the flesh of animals killed when overdriven or in an exhausted state, is liablè to produce symptoms of irritant poisoning.

Pork.—Perhaps there is no kind of ordinary food so liable to give rise to accidents of this description as pork. Several cases were published by the late Dr. Mac Divitt, tending to show the noxious effects of this substance. One of these, it appears, led to a strong suspicion of criminal poisoning.

The patient, a young man, conceived that he had been poisoned; and accused a woman with whom he lived, and her mother of having administered something deleterious to him in tea, which he had taken three hours previously. The following were the symptoms under which the patient laboured:—there was an acute burning pain in the umbilical and epigastric regions, attended with almost constant vomiting of a dark-coloured fluid, which contained pieces of half-digested meat. He complained also of a dry, burning sensation in his throat. His pulse was weak and faltering, his extremities cold, and a cold perspiration covered his face and head. These symptoms, coupled with the well-known bad character of the young woman and her mother, led the medical attendant to suspect that poison had been administered. An emetic was immediately exhibited, which brought up some pieces of half-digested meat, with immediate relief to the pain in the epigastrium. The bowels were then acted on; and the pain in the umbilical region, which had begun to extend itself over the whole of the abdomen, was also speedily removed. On the next morning, the patient was quite well.

In investigating the particulars of the case, Dr, Mac Divitt felt satisfied that these alarming symptoms must have been due to the effects of pork, since they bore the strongest resemblance to other cases of that kind which he had witnessed. He ascertained that the man had had for dinner, that day, boiled pork, and the broth made from it. The pork had been salted five days: and the man had partaken of it once before, without experiencing any ill-effects. He had dined at one o'clock on the last occasion, and the violent symptoms had appeared *seven hours and a half* afterwards. The man referred them to the tea which he had taken three hours before. The young woman and her mother, who had dined with him, were not in the slightest degree injuriously affected.

In relation to this case, it may be observed, that had the patient died under the attack—an event which, from the severity of the symptoms, could not have been regarded as very surprising—the two females might have been tried on a charge of murder. The notoriously bad characters of the accused—the terms of disagreement on which they had for some time before lived with the young man—his own conviction that they had administered poison to him—were facts which taken together with the nature of the symptoms, might have appeared to a jury to form a strong and well-connected chain of circumstantial evidence. The negative results of experiments instituted with a view to detect the presence of poison in the stomach after death, might not have been deemed conclusive of the innocence of the accused. At any rate, the fact, that the symptoms alone could not have been distinguished from those of irritant poisoning, proves sufficiently the importance of knowing that such a simple cause is capable of producing them.

Five cases precisely similar are reported, They were all relieved by the free use of emetics and purgatives. Dr. Mac Divitt observes, in summing up the results of his observations, that nothing remarkable could be discovered in the sensible properties of any piece of pork, the use of which had been followed by injurious consequences. The symptoms have resulted from boiled, as well as from roasted pork, but more frequently from the latter. He never met with a case in which they had arisen from the use of bacon or of pork which had been salted for a longer period than three weeks. He conceives that it is chiefly, if not altogether, to the fatty parts of the pork that the pernicious effects are to be attributed. The symptoms may display themselves at any period *within thirty hours* after the meal at which the pork has been eaten; and it is rare that they commence until *three hours* have elapsed. The most careful chemical analysis has entirely failed in detecting any poisonous matter in pork, under these circumstances.

It will be observed, that there are some peculiarities about these cases of poisoning by pork, reported by Dr. Mac Divitt. The symptoms were decidedly similar to those of irritant poisoning. Some hours (seldom less than three) elapsed after the meal, before they made their appearance: but the most remarkable points are—1. That the food which exerted this poisonous action had been previously eaten by the same individual with impunity. 2. That other parties, who partook in common of this food, did not suffer any ill effects. It is difficult to suggest any explanation of these anomalies; they led Dr. Mac Divitt to adopt the view, that the pork could not have been in itself poisonous, but that it had been rendered noxious during the process of chymification. If this explanation be admitted as correct, this must be regarded as another mode in which animal food may become poisonous to a human being. I am not aware that there are any other facts corroborative of this opinion; they can neither be referred to habit, nor easily to idiosyncrasy.

Bacon.—The effects of bacon in an unsound state are not very well known; but the following case, which occurred in London in 1836, excited some attention at the time, and the circumstances underwent a full investigation.

An inquest was held on the body of a girl, aged 15, who was alleged to have died from the effects of unsound bacon. The father of the deceased stated, that he and his family, including the deceased, had had the bacon for dinner with greens: he thought that it had a nauseous taste: he ate very little of the fat, and none of the lean. The deceased ate very heartily, especially of the lean part of the bacon. Soon after dinner he felt a burning sensation in his throat, and a disposition to vomit. About an hour after dinner, his son was seized with pain in the stomach, and vomiting. The deceased, who left the house shortly after dinner, returned about five o'clock, complaining of dreadful headach, and burning at her stomach; she swallowed some tea, and went to bed; but nothing was ejected by vomiting. About this time, another child, aged 8, was attacked with similar symptoms. There were two other children, who had not partaken of the bacon, and they were not affected with any illness. He gave his children some calomel pills, which he procured at a druggist's but no medical assistance was called in until after the lapse of a fortnight. The deceased was then removed to Westminster Hospital, where she gradually sank, and died about six weeks after the meal at which she had taken the alleged unwholesome food. The chief symptoms when she was admitted into the hospital, were pain in the epigastric region and profuse diarrhœa. She became much emaciated before her death. On a post-mortem examination, the lining membrane of the stomach and duodenum were found slightly reddened. In the lower part of the small intestines, as well as in the colon, irregular patches of ulceration were met with. One ulcer had perforated the coats of the ileum, near the cæcum; and this had led to the effusion of the contents of the intestine, and had given rise to peritonitis, affecting the serous covering of the lower half of the abdominal viscera. The bacon had been thrown away. Every one who partook of it suffered; but no one so seriously as the deceased. It was observed, on cutting it open, to have a nauseous disagreeable smell, but not at all like that of putrid meat. Upon further inquiry, it was found that the bacon had been sold by a regular dealer. No complaint had been made of its bad quality by others; but he admitted that the piece sold to this family was of inferior quality, compared with the other portions.

The bacon, in this case, seems to have acted as an irritant poison; and the symptoms of irritation were manifested very speedily after the meal. The circumstance of many persons having been attacked simultaneously with similar symptoms, from the use of the same article of food, in which there was not the least reason to suspect the presence of any mineral irritant, appears to me to prove that the symptoms must, in this case, be referred to the noxious properties of the food itself. It does not seem that putrefaction had taken place, but it is not improbable that the same change had occurred in it which is occasionally observed in the fatty parts of German sausages. It was stated in evidence, however, that the deceased had partaken more of the lean than of the fat; so that it is probable the production of the poison, by fermentation, had extended throughout the whole substance of the bacon. Another fact appears, that the deceased ate more of this bacon than the other members of the family; and this will account for the severity of her illness and death. It does not appear, however, that her death was occasioned by the direct action of the noxious food: it was rather an indirect result, after a long period, depending upon ulceration and perforation of the viscera. There are many points in which this case resembles those of poisoning by veal. (ante, p. 448.) Ulceration of the intestines was found in both instances. This process may be set up from the continued action of any irritant, whether belonging to the mineral, vegetable, or animal kingdom; but it is only likely to occur where life is for some time protracted after the cause of irritation has been introduced into the alimentary canal. It is proper to remark that these cases establish in the affirmative, what has been often a subject of inquiry; namely, that the cooking of noxious animal food does not counteract or destroy its poisonous properties. More recently, three cases of poisoning by bacon have been reported by Mr. Toynbee to the Medico-Chirurgical Society: here, however, with the symptoms of irritation, there was some affection of the nervous system. A gentleman, æt. 35, partook three times of tainted bacon, and it was only on the afternoon of the third day that he felt himself becoming dreamy, that his imagination was disordered, and that there was almost an entire loss of memory. There was slight diarrhœa; pulse slow and weak; the hands were cold and clammy; his intellect was unaffected. These symptoms disappeared in three or four days. In the second case, a gentleman, after eating the bacon once, experienced sickness, depression, and chilliness. The next day there was a burning sensation in the throat, acute abdominal pains, confusion of ideas, and dimness of vision. There was also diarrhœa, with cold clammy perspiration. In the third case, the patient having partaken of the bacon at breakfast three times, felt nothing until the evening of the third day, when he was seized with nausea, faintness, and severe spasmodic pains. There was diarrhœa, with disturbance of the mental faculties. He only recovered after three or four days. (Med. Gaz. xxxvii. 470.)

Pestis Bovina.—In some instances the poisonous quality of the food is clearly referable to the disease with which the animal was affected when killed. This is especially the case in the epizootic disease called carbuncle, frequently prevailing to a great extent among cattle on the Continent. The following case appeared about a year since in the Annali Universali di Medicina, and has since been published in the Edinburgh Medical and Surgical Journal (lviii. 262.)

A heifer, which had two carbuncles on the buttock, was killed, and its flesh sold. It appears that about sixty persons partook of this, as food; and all were seized with the following symptoms—giddiness, trembling, shivering, violent cramps in the abdomen and limbs, vomiting and purging of a green bitter matter, intense thirst, sinking of the countenance, and delirium. The tongue was observed to be red at the tip, and furred at the base. These symptoms were severe in proportion to the quantity of flesh of which each person had partaken. With one exception, all the patients recovered under the use of very simple remedies. In the solitary case that proved fatal, the symptoms were not different in character from those above described, but they were much more severe. The prostration of strength increased rapidly; there was loss of voice, and a soporific state ensued. This patient died on the second day after admission into the hospital. On a post-mortem examination, the body was found to be much emaciated; and there were livid spots scattered over the skin, especially over the lower extremities. The veins of the dura mater were filled with blood, and the spinal marrow was somewhat softened. In the abdomen, the liver had a tendency to softening, and the spleen was diminished in size. There was submucous ecchymosis, occupying about two-thirds of the greater curvature of the stomach; a similar ecchymosis, near the cardiac orifice; and spots of the same character were found at intervals over the whole surface of the intestines.

In this report, it is not stated at what time the symptoms came on after the meal, nor how long a time elapsed before death, in the case of the person who was poisoned. It is probable that the patients were speedily conveyed to the hospital, and therefore that death occurred in about forty-eight hours. In this disease, it is said the flesh of the animal is rendered so poisonous, that the mere handling of it is liable to occasion formidable symptoms. Both the solids and liquids of the animal appear to become poisoned, under its influence. (Guy's Hospital Reports, April 1843: see also Ann. d'Hyg. 1829, ii. 267; 1834, ii. 69.)

Partial decay.—There is no doubt that partial decay may render unwholesome and injurious, the flesh of the most healthy animal, and it is by no means improbable, that among the poor of large cities, the secret sale of decomposed and unwholesome meat is a very frequent cause of disease and death. What the nature of the poison is, which exerts such a virulent action on these occasions, we are quite unable to determine. Liebig imagines that it is owing to the production of a fermenting principle, and that it operates fatally by inducing a kind of fermentation in the animal body. It has been said that the symptoms of irritant poisoning, produced by animal food, seldom appear until five or six hours after the meal. This may be generally true; but in certain instances it has undoubtedly happened that the symptoms have come on in from a quarter to half an hour after the taking of the noxious food,

Flesh poisoned by food.—In the number of the Edinburgh Medical and Surgical Journal for July 1844, it is observed, that all the effects of poisoning may be produced, without the animal having been apparently affected with any disease. In some extensive districts of North America, to the west of the Alleghanies, the herbage has no injurious effect upon the animals which are there pastured; but their milk and flesh, when used as food, are poisonous to man. The disease produced by the use of the flesh or milk of animals fed in these districts, is known under the name of the milk-sickness, or "*trembles*." On account of the prevalence of this malady in a particular locality, which is generally distinctly circumscribed, the early emigrants were often compelled to seek another; and those who now venture within the boundaries of an infected district, are constrained, as a condition of their residence, to abstain from the flesh of the cattle living within the same limits, as well as from the milk and its preparations. It appears from the late report of Drs. Hosack, Post, and Chilton on this subject, that in some of these infected districts, the inhabitants, with a recklessness of human life which seems incredible, carry the butter and cheese which they themselves dare not eat, to the markets of the towns west of the Alleghanies, and that thus there are frequently produced symptoms of poisoning and even death, for which the medical attendant cannot account;—he is, perhaps, induced to consider it as some new or anomalous form of disease. It is also stated, that the cattle from these districts are sent for sale in great droves over the mountains; but, in order to deceive the buyers as to the place whence they come, they bring them to New York by a southern route, and style them "southern cattle." The flesh of these animals produces, in those who make use of it, symptoms of aggravated cholera morbus. The viscera of the animals are often found diseased, and the livers almost invariably so. Owing to the symptoms which have followed the use of the beef, butter, and cheese thus poisoned, the municipal authorities of New York caused a medical inquiry to be instituted into the matter with a view to prohibit its sale.

[See Am. Med. Recorder, vi. 257. Am. Journ. Med. Sci. 1841. New York Journ. of Med. 1843.—G.]

Putrid eggs.—Dr. Marshall has reported a case in which four persons were seized with well-marked symptoms of poisoning after eating for their supper some *eggs* which were *decomposed*. One man appeared to be in a state of coma, from which it was difficult to rouse him: his face was livid, his lips blue, his eyes open and fixed, limbs flaccid, and respiration slow. His wife, brother, and one of his sons were affected, although in a less degree, with similar symptoms—complaining of vertigo, weight and pain in the head, pains in the limbs and disinclination to move. It appears that the eggs which had been eaten in a pudding had a disagreeable and a slightly putrid smell, and the whole of these persons were taken ill soon after the meal. The symptoms were not those of irritation, but of narcotism: they all recovered. (Brit. and For. Med. Rev. xvii. Jan. 1840, p. 267.)

[Many cases of poisoning have occurred in the United States from the flesh of the pheasant (TETRAO UMBELLUS.) This bird, during the winter season, is said to feed on the berries and leaves of the laurel (KALMIA,) and that in consequence its flesh becomes deleterious, and many facts have been adduced, which are considered to corroborate this opinion, the most striking of which, is the presence of the leaves in the crops of the birds. This explanation does not appear satisfactory, and I am inclined to attribute the poisoning to some change in the flesh itself, as the symptoms are almost identical with those induced by the diseased meat of other animals, and still more from the fact, that these cases of poisoning are rare, and yet during the winter almost all these birds feed on the laurel, especially if the ground is covered with snow, and other food cannot be procured.—G.]

POISONOUS MILK.

This animal secretion has been observed to acquire spontaneously poisonous properties in certain cases. The following instance is reported by Dr. Krummacher. A woman while suckling her child became violently excited by the loss of some articles which had been stolen from her. She gave her child the breast while in a state of violent passion. The child at first rejected it, but subsequently took a quantity of milk. Soon afterwards, violent vomiting supervened. In the course of some hours the child took the other breast, when it was attacked with violent convulsions, and died in spite of medical aid. (Casper's Wochenschrift, 22d March, 1845, p. 204.) Cases in which the milk of a wet-nurse has produced serious symptoms in infants, may be found recorded in most works on midwifery.

Symptoms of irritation have been also observed to follow the use of *Cow's milk*. In some instances, these effects have been traced to the milk having acquired a poisonous impregnation by contact with zinc or copper, in which case the lactic acid of the milk, would tend to form lactates of these metals. On other occasions, it would appear as if the secretion had really acquired a specific poisonous action. Eight persons were seized with pain in the abdomen, thirst, diarrhœa, vomiting, and fever, after having partaken of some fresh cow's milk. One, who took the largest quantity, did not recover for several days; another, who did not partake of the milk, had no symptoms. The milk, it appears, had been drawn as usual in a wooden pail, and afterwards placed in an earthen pan. On the day following, two persons were seized with severe pain and diarrhœa, after drinking milk drawn from the same cow. Poisoning was suspected, and an inquiry was instituted. It was remarked from the first that the cow appeared unwell, and that the udder was gorged. The milk was analysed, but no trace of any poison, mineral or vegetable, could be discovered either in it, or in the matters vomited. The injurious effects of the milk were ascribed by M. Chevallier either to a diseased state of the animal, or to the milk having been poisoned by some noxious vegetable taken in its food.

Another opinion was given, that, as in the human female, the milk might have acquired noxious properties, by spontaneous changes, after its secretion, and while in the lactiferous vessels.

The facts could leave no doubt that the symptoms analogous to irritant poisoning were dependent on the milk, yet no peculiarity of colour, odour, or taste, could be perceived about it; and a portion of the milk of the diseased cow was given to some pigs, without producing any injury. It is not at all improbable that some difference in the physical constitution of this poisonous milk, may be hereafter detected by the use of the miscroscope. (See Journal de Pharmacie, Dec. 1846, 425; also Ann. d'Hyg. 1846, i. 139.)

It is generally admitted that milk may become poisoned when the cow feeds upon *hyssop* (ante, p. 414,) *spurge* (ante, p. 413,) and other irritant vegetables; and this form of poisoning is well known to occur in other cases in which the cause is not so apparent (ante, p. 452.) In the Dictionary of Natural History a case is related, where a patient was advised by his medical attendant to drink the milk of a cow fed on hemlock. The animal became emaciated, lost its milk, and fortunately for the patient died from the effects of the poison, or it is not improbable that he might have fallen a victim to this plan of treatment. This secretion easily undergoes changes, according to the food of the animal. It it rendered bitter when the cow feeds on wormwood (*Artemisia absinthium*,) on Sow-thistle (*Sonchus alpinus*,) the leaves of the artichoke (*Cynara scolymus*,) and its taste is affected by the cabbage, carrot, and all strong-smelling plants. The milk-sickness of America has been already described: the effects extend to butter, cheese, and all articles of food prepared with milk (ante, p. 453.)

At Aurillac, in France, sixteen persons were seized with violent sickness after having drunk the milk of a *goat*. The animal became indisposed in two days, and died on the third day, with symptoms of irritation of the alimentary canal. This poisonous action of milk has been often referred to the animal having eaten the *Euphorbia esula*, but nothing certain is known on the subject. It is singular that the animal poison of rabies should be sometimes transmissible by the milk (see Med. Gaz. xxv. p. 160; also APPENDIX) and at other times not (see p. 459, post.)

CHAPTER XXXIV.

ANIMAL POISONS CONTINUED—POISONING BY INOCULATION—POISON OF GLANDERS—EFFECTS OF MORBID AND PUTREFIED ANIMAL MATTER—RABIES CANINA—HYDROPHOBIA—ORIGIN OF THE DISEASE—PERIOD OF INCUBATION—SYMPTOMS—POST-MORTEM APPEARANCES—TREATMENT—PROPERTIES OF THE SALIVA—SYMPTOMS OF RABIES IN THE DOG—POISON OF VENOMOUS SERPENTS—SYMPTOMS AND APPEARANCES—TREATMENT—VENOMOUS INSECTS—SCORPION—SCOLOPENDRA—SPIDER—ARGAS PERSICUS—BEE—WASP—HORNET.

WE have hitherto considered the action of Animal poisons as they affect the system after they have been taken into the alimentary canal. There are two other ways whereby these poisons may destroy life—through the respiratory organs, as by the inhalation of the foul and noxious effluvia arising from decomposed animal matter; and, secondly, by inoculation, as by the introduction of poisonous matter into a wound in the skin. The first of these modes of poisoning will be considered in speaking of suffocation by poisonous gases;

the second, or that which refers to poisoning by *inoculation*, demands a slight notice.

Of the effect of such specific poisons as those of the Small-pox, of vaccine lymph, and of the matter of the plague, nothing need be said, as these subjects are more appropriately treated in works on practical medicine.

DISEASED AND PUTREFIED ANIMAL MATTER.

Certain diseases to which animals are liable, such as PESTIS BOVINA, GLANDERS, and RABIES, vitiate the blood and fluids of the body, and act as poisons on man when introduced through a wound or even by direct application to an ulcerated or any other absorbent surface, *e. g.* the mucous membrane. The *Malignant pustule* is a disease induced by this kind of poisoning. It is marked by diffused inflammation of the cellular membrane—the production of phlyctenæ, and abscesses terminating in gangrene and death. A disease which affects horses, known under the name of GLANDERS or FARCINOMA, has been frequently transmitted with fatal consequences from these animals to man. In pustular glanders, the nostrils of the horse discharge a sanious purulent matter in which the animal poison resides. In the more severe cases, the mucous membrane becomes gangrenous, and the disease runs through its course with fatal rapidity. The chronic state of this disease in the animal, is known under the name of *Farcy*. The period of incubation of this poison in the human subject, is about four or five days. There are rigors and severe pains in the limbs—the inoculated part becomes inflamed, and suppurates, the inflammation extends to the absorbents, the lining membrane of the nose and the trachea; there is, at the same time, a copious discharge from the nostrils. A pustular eruption spreads over the body, and gangrenous abscesses show themselves. There is diarrhœa, with bloody stools, and a dry black tongue:—delirium and stupor precede death. (Gregory's Practice of Medicine, 239.) It rarely happens that the patient recovers:—of fifteen cases, it was observed that only one survived, and two-thirds of the recorded cases proved fatal before the seventeenth day.

Inoculation with animal matter in a *putrefied state* is also liable to produce inflammation of the cellular membrane, enlargement of the glands, and suppurative fever. Cooks and butchers are exposed to these accidents, and in some cases no kind of treatment has sufficed to avert a fatal result. The punctures received during *dissection* are often attended with severe inflammation of the absorbents, swelling of the glands, abscesses, and irritative fever, passing to low typhus, proving fatal. The period of incubation in these cases is from twelve to forty-eight hours. Pain and swelling first appear in the wound, and then extend to the absorbent vessels. Death takes place in from three to seven days: in one instance it occurred in forty hours. These effects have not been in all cases traced to a *putrefied* state of the body and the inoculation of *decomposed* animal matter. (See Lancet, April 17, 1847, p. 421.) On the contrary, it is often, in recent subjects, before putrefaction has commenced, that the most serious effects are produced by an accidental puncture received in dissection. In these instances, the effect can only be ascribed to the generation by disease of a specific animal poison. It has been observed that those punctures which are received in the dissection of subjects, who have died from peritoneal inflammation, although the bodies may be recent, are very liable to be followed by the most serious consequences. The bodies of females that have died of puerperal fever have thus furnished the most deadly poison. I have known death to take place in more than one instance from slight wounds received under these circumstances. (See also a case by Mr. Colles, Med. Chir. Rev. vii. p. 307; and Chelius's Surgery, Amer. edit.

vol. i. p. 386.) Whenever there is an abrasion of the fingers; it is always dangerous to allow the hands to come in contact with morbid or diseased fluids. A case in which poison was thus communicated by the discharges attending the delivery of a female will be found in the Medical Gazette (vol. xxxvii. 1088.)

Whatever difference of opinion may exist respecting the presence of an animal poison in dead bodies generally, there can be no doubt of its existence in cases of death from inflammation of the serous membranes, as the danger from inoculation is observed to be great in proportion as the inspection of the body is recent.

The best plan of *treatment* in inoculation by animal matter, is to apply a ligature above the wound, so as to prevent the return of the blood,—to enlarge the opening, wash the wound in warm water, and allow it to bleed freely. The application of caustic is of questionable utility. Absorption does not appear to take place very rapidly in these cases: but the rapidity of thisprocess is found to be greatest when the stomach is empty.

One animal poison, arising apparently from a vitiated state of the fluids, requires to be especially noticed, namely, RABIES CANINA.

RABIES CANINA. HYDROPHOBIA.

The frequency of death from this cause, and the fact that fatal cases by hydrophobia are invariably made a subject of inquiry before the coroner, render it necessary to make a few observations on the effects of this remarkable animal poison.

Hydrophobia, or the dread of water, is the main pathognomonic character of the disease produced in the human subject by the poison of rabies, although this symptom may be present in other morbid states of the nervous system. It is singular that the symptom by which the poison thus manifests its presence in the human subject, it not met with in the animals which impart it; for they have been observed to drink fluids without repugnance until within a short period of their death. Mr. Youatt, who had much experience on this subject, never witnessed hydrophobia (*i. e.* the dread of water) in any animal: it was observed only in man. Rabid dogs commonly have an insatiable thirst and drink water very readily.

Rabies, or the disease which generates the hydrophobic poison, occurs *spontaneously* in the dog, fox, wolf, jackal, and cat. It is more commonly witnessed in the dog-kind (genus Canis,) but the disease in man is said to be produced more uniformly and rapidly from the bite of a rabid cat, than from that of a rabid dog. The poison may infect and produce fatal effects on man and herbivorous animals, as the horse, the ox, the sheep, and the pig; but there is no authentic instance known of its having been communicated by these animals to others, nor has it ever been observed to arise *spontaneously* in man or in herbivorous animals; although Mr. Youatt states that the saliva of a rabid ox produced rabies in a dog inoculated with it. The poison is transmitted through the *saliva:* but in what way the secretion is rendered poisonous by disease in the animal, or what is the exact nature of the poisonous agent, it is impossible to say. Some pathologists think that the poison really exists in the mucous secretions of the trachea and bronchia, and that it becomes merely mixed with the saliva. In the human subject, the local effects in the part bitten are commonly slight, but in an interesting case reported by Mr. S. Wells of Malta, where a woman was bitten by a cat, the hand swelled greatly after the bite, the pain and swelling extended to the shoulder, and she was obliged to carry her arm in a sling for a fortnight. This lady was seized with the usual symptoms in about four months, and died. The cat had been bitten by a rabid dog twenty-two days previously. (Med. Gaz., vol. xl. page 112.)

There is some difference of opinion, as to whether the disease can be conveyed without a wound or abrasion of the skin. A wound, however trivial, such as an abraded pimple, undoubtedly suffices for inoculation, provided the saliva of the animal has come directly in contact with it. The Hon. Mrs. Duff died of hydrophobia from this cause. She had been in the habit of allowing a French poodle to lick her face. It licked the abraded surface of a pimple, and led to inoculation with the saliva,—producing subsequently hydrophobia and death. This is not only a most disgusting, but a dangerous practice: as it is impossible to say whether the saliva of an animal be or be not in a morbid condition. The fine skin of the lips will even, in an unabraded state, absorb the poison. Dr. Colles met with the case of a young girl, where the contact of the saliva of a rabid dog thus led to a fatal attack of hydrophobia. (Lectures on Surgery, i. 83.) Applied to the rough unbroken skin, the poison might have been without effect;—at least no instance is known of hydrophobia having been thus produced. When the inoculation takes place by a bite, the slightest abrasion of the cuticle will suffice for absorption. If the tooth should have perforated articles of clothing before meeting the skin, it is possible that the mucous secretion may have been entirely wiped from it. This will explain why among a number of persons bitten by the same animal, many will escape, while one or two may suffer from hydrophobia, and all other animals bitten by it will have rabies. According to Professor Colles, the number of escapes in the human subject, is as fifteen to one attacked. This circumstance also accounts for the apparent success of numerous so-called preventives or antidotes:—the poison may not really have been introduced by the penetration of the tooth. The hydrophobic poison must be of a most subtle nature; for, like that of venomous serpents, the minutest quantity will suffice to produce the disease.

Period of attack.—The period of *incubation* in the human subject appears to be tolerably well settled. The disease makes its appearance most commonly in from *thirty to sixty* days after the introduction of the poison. Dr. Gregory places the average at forty days or six weeks, after which the chances of escape are greatly increased. The shortest period at which the disease manifested itself, was twenty-one days: and the longest twelve months (in a case of Mr. Cline's;) although in some instances it is stated not to have appeared until eighteen months after the bite. (Colles, op. cit. 85.) In the dog and cat, the earliest period of attack after the bite was fourteen days; the average time is five or six weeks.

As a proof of the remarkable nature of the hydrophobic poison, there is often no local irritation during this long period in the part bitten; although it can hardly be said to be removed by absorption, because on the commencement of the symptoms, pain is usually felt at the part, with numbness extending up the limbs in the course of the nerves. Unlike the animal poisons already considered, the absorbent vessels do not become inflamed or in any way affected. The bite commonly heals readily, and without any unpleasant symptom.

Symptoms in man.—At the commencement of the disease, the cicatrix of the wound sometimes becomes inflamed—a creeping sensation as of coldness or numbness extends from it up the limb to the trunk—the patient becomes restless and irritable; his senses are morbidly acute; he dislikes the smell of any familiar substances, and the reflection of light from polished surfaces; his pupils are dilated, and his countenance expressive of timidity and great anxiety. He sits in a cowering posture and shudders when any one approaches him. The patient has been observed to shrink sometimes under the bed-clothes in the most dreadful state of fear. The first symptom in the Duke of Richmond, was that early in the morning his valet found him alarmed at the appearance of some trees which were near to a window of the room where he slept, and which he insisted were people looking in. The attack which proved fatal to this nobleman in Canada in 1819, is attributed to his having been bitten in the thumb by a tame fox, six or seven weeks before his seizure. The symptoms above described are generally preceded by headach, languor, and depression of spirits. The respiration is hurried and gasping; there is a suspicion of those about him, and the patient feels a sense of oppression in the epigastrium. There is thirst;—the saliva becomes increased in quantity, is very viscid, and escapes from the mouth in a frothy state. It is this which renders breathing somewhat difficult, increasing the gasping of the patient, and it has thus given rise to the idea of his barking like an animal. Notwithstanding the thirst, he is quite incapable of drinking:—the sight of fluid commonly occasions spasms of the throat and diaphragm, with violent convulsions. Owing to the extremely irritable and excited state of the patient, a current of air, or the noise of water or liquids being poured from one vessel to another, brings on an attack. There is commonly no attempt to injure those about him. There is no madness, the mind generally remains clear until the last, and there is very seldom delirium. Increased excitement and not debility appears to be the main feature of the disease. The patient commonly dies exhausted by the frequent spasmodic attacks in from two to five days. Very often death takes place in less than two days, during a violent convulsive fit. The disease is uniformly fatal:—there is no authentic instance of recovery known. Mr. Youatt gives the following summary of its duration. In man it has run its course in twenty-four hours, and rarely exceeds seventy-two; in the horse, from three to four days; in the sheep and ox, from five to seven; and in the dog, from four to six days.

Post-mortem appearances.—All that has been observed on inspection was a reddened or inflamed condition of the mucous membrane covering the epiglottis, larynx, pharynx, and of the cardiac orifice of the stomach. In one case Mr. Curling found a very enlarged condition of the mucous follicles of the pharynx and larynx. (Med. Gaz. xviii. 736.) There are no well-marked morbid appearances of the brain or spinal marrow. It is said that the medulla oblongata has been found inflamed, but in other instances there has been no change.

TREATMENT.—The treatment of hydrophobia by every variety of agent has hitherto proved unsuccessful. Even the new and powerful agent, ether-vapour, failed to prevent the occurrence of the spasmodic attacks. (Med. Gaz. xl. 112.) It is to the *prevention* of the disease that attention must be chiefly directed: and experience shows that the effectual and speedy excision of the bitten part, wherever situated, is the only measure which promises a hope of success. Caustic may be employed with it, but it is entirely useless without excision. Professor Colles has known this plan to succeed when the operation was not performed until seven or eight days after the bite, and it should even be tried after much longer periods than this: for there is reason to believe that the poison, if at all, is only very slowly removed by the absorbents. Professor Colles proved the efficacy of excision in one remarkable instance. Three persons were bitten by the same dog at the same time, two of them suffered the part to be cut out, and they escaped—the third refused to submit to the operation, had hydrophobia and died. (Op. cit. 88.) A cupping glass, or other partially-exhausted vessel, may be placed over the bitten part, until excision can be resorted to, or a ligature may be applied above the wound. Perhaps hereafter some means may be discovered of counteracting this by another animal poison of a milder kind, just as the vaccine disease counteracts small-pox.

Nature of the poison.—The nature of this poison, as it is generated in the dog, is quite unknown. It is very probable that chemical analysis would show the existence of no important difference in the saliva of the animal, whether healthy or diseased. Dr. Wright believes that the secretion contains no animal poison, but that the saliva itself possesses in an undue degree, the physiological activity which is natural to it. (Lancet, April 20, 1844, 124.) We have not the least knowledge of the morbid condition of the body, requisite for the production of the hydrophobic poison. The animal may be to all appearance healthy, and yet the saliva will possess this poisonous property. Hydrophobia is said to have been caused by the bite of a dog, during the excitement produced by its fighting with another. One among several instances is quoted by Dr. Colles, in which the disease was communicated by the bite of a dog not apparently affected with rabies. (Op. cit. ii. 89.) That it is a specific poison is proved by many facts,—among others by the following, on the authority of Professor Coleman. A sow was bitten while pregnant, and the whole litter of pigs were subsequently seized with rabies. (Med.-Chir. Rev. vii, 236. For similar cases see Med. Gaz., Oct. 25, 1839.) This is analogous to the transmission of arsenic from the mother to the fœtus in utero. It is singular that, among the precursory symptoms, pain should be perceived in the part bitten. This would appear to show that there must be a peculiar local, as well as a general effect on the system. A question of interest in relation to the transmission of the poison through different secretions may be noticed:—it appears to be specially transmitted through the *saliva.* A child was fed with goat's milk up to the time at which the animal was seized with rabies; but no evil effects followed. A cow had been bitten by a dog affected with rabies. As no symptoms immediately appeared in the animal, the milk was used to feed a child, aged fifteen months. On the day on which the milk was so employed, the animal manifested the usual signs of rabies. The parents were very anxious about their child, but no symptoms indicative of hydrophobia appeared. (Ann. d'Hyg., 1846, i. 146.) Mr. Steele has, however, reported a case in which the milk of ewes affected with rabies communicated the disease to the lambs (see APPENDIX; also Med. Gaz. xxv. p. 160.)

Symptoms of rabies in the dog.—In the first stage of the disease the animal appears sick, dull, and peevish, but becomes playful at intervals. He does not appear to know those to whom he has been most attached, and his habits are completely altered. He snaps at the air as if at insects, drinks his own urine and dung, and swallows dirt, straw, and all articles within his reach. He roams about, running in an irregular manner, with his back arched and his tail drooping, though not drawn beneath the body: he runs or swims through water without difficulty. In his progress he avoids other dogs, not generally going out of his way to bite them; although he will snap at them and bite them if he happens to come near them. It is a remarkable fact that all other dogs avoid him. The voice is altered: the bark is between the ordinary bark and the howl, and ends with a short peculiar howl. The animal does not refuse to drink water even to the last, nor does he foam at the mouth as is commonly supposed. (See Chelius's Surgery, Am. ed., vol. i. p. 399.) The disease in the dog is, therefore, not characterized by *hydrophobia*. Dogs are affected by several diseases resembling rabies: they should in these cases be secured and watched. The animal wastes daily in strength, and generally dies suddenly in from six to eight days after the first attack. After death, the fauces generally exhibit traces of inflammation, and in and about them various substances are found, which the animal has not had the power to swallow. There is an inflammatory redness of the larynx, occasionally extending to the trachea. The stomach is also more or less reddened, especially about the rugæ; and all kinds of extraneous substances, in one mingled mass, may be found in this organ. Sir Astley Cooper in one instance found blood effused between the mucous and muscular coat. No particular appearances have been remarked in the brain, except in some instances inflammation of the membranes.

The dog is subject to rabies at any period of the year; according to some, it is dependent on bad food, changes of heat and cold, and unsatisfied sexual desires. Rabies is popularly regarded as being more frequent in summer than at other seasons: but if it depended on heat of climate, it would be very frequent in the tropics, whereas it is much more rare in hot, than in cold latitudes. The poison of venomous serpents which abound in hot climates is not more destructive to human life than the poison of rabies:—the one is a natural and the other a morbid secretion. For some remarks on the subject, see Med. Chir. Rev. vii. 236; also Lancet, May 1, 1847, 464.)

It is an important question whether enraged animals may not have this poison generated in the saliva. Several instances are on record which certainly bear out this view. A man æt. 30, was admitted into St Bartholomew's Hospital, March 20, 1828. About ten weeks previously, he had been bitten by a cat deeply on the ring-finger of his left hand and upon the calf of his leg, while he was attempting to save one of his children. This child was likewise bitten, but no symptoms had shown themselves. It is not stated that the cat was affected with rabies. The man refused to have the wounded part excised. The wound healed readily, and he thought no more of it until the 17th March, when he felt a stiffness in his left hand, followed by pain shooting up his arm. The pain rapidly increased in severity until the day of his admission. He then tried to swallow some wine and water; but he had no sooner taken a little than he sprang from his bed in strong convulsions. When brought to the hospital the same evening, his look was wild and anxious, eyes staring, pupils dilated. He was calm and rational; pulse 80, and full; skin moist. The saliva adhered to his lips, producing the appearance of frothing, and it was with great difficulty removed. He was rather thirsty, but the attempt to swallow produced spasms of the muscles of the throat and of the diaphragm. These were even brought on by any one passing rapidly near him. He died on the 21st, the fourth day from the first appearance of the symptoms. On inspection, there were marks of inflammation at the back of the fauces, not extending to the trachea and œsophagus, but strongly marked in the bronchi and their ramifications. The arachnoid membrane was opaque, and the cerebral vessels congested:—similar appearances were found in the spinal cord, but they were not very strongly marked. The marks of the wounds were scarcely discernible. (Med. Gaz. i. 517.) The bite of an enraged dog is very likely to cause hydrophobia. (See p. 459.)

POISON OF VENOMOUS SERPENTS.

According to Schlegel, out of two hundred and sixty-three species of serpents, fifty-seven only are venomous, and of this number there is only one, the COMMON VIPER (*Vipera Berus*, *Coluber Berus*,) which is met with in northern latitudes. This animal is found in the central parts of Europe, including England and Scotland. In southern Europe it is replaced by the *Vipera Aspis* or Aspic. The viper, as it is found in England, is about eighteen or twenty inches long, of a dusky-brown colour on the back, along which there extends a chain of irregularly rhomboidal dark brown spots from the head to the tail. It is of a lighter colour underneath.

The gland which secretes the poison in venomous serpents is situated near the orbit:—it communicates with a receptacle or sac which forms anteriorly a wide duct into which the base of the tooth or poison-fang is inserted. The fang has a curved form, is exceedingly sharp at the point, and when not in use, lies folded along the upper jaw. In the rattle-snake the poison-fang is more than an inch long—there are commonly on each side one developed and one rudimentary. The base of the tooth is perforated, and a fine canal extends from it, terminating on the anterior or convex surface of the tooth by a fine oblique aperture, so that the sharpness of the point is not interfered with. When the animal is excited, the muscular apparatus which raises the tooth, compresses the sac containing the poison, and jerks a small quantity through the canal of the tooth into the wound.

The effects of this poison vary according to the size of the serpent and the size of the animal bitten. Duméril states, that the 1-250th of a grain of the poison of the viper will suffice to kill a small bird. (Elemens des Sciences Naturelles, ii. 235.) The poison is in small quantity and slowly secreted—the sac of a viper rarely contains more than a grain and a half. It is therefore soon exhausted, and after the animal has bitten two or three times, the poison appears to lose its virulent power. The deeper the fangs pierce, and the more completely the part bitten is included within the animal's jaws, the more dangerous is the bite. In winter the poison is less active than in summer.

The effects of the poison of venomous serpents, are most strongly manifested when it has been introduced through a wound, and the larger the quantity the more serious the symptoms. The slight injury done to a man by the bite of a viper, appears to be owing to the very small quantity of poison ejected. According to Schlegel, whilst a single bite will kill a small mammiferous animal or a bird, it requires the bite of three or four vipers to kill a horse or an ox: hence it happens that this poison is rarely fatal except in very young subjects.

Symptoms and appearances.—The more rapidly the symptoms appear, the more dangerous they are likely to be. Both fangs of the reptile commonly enter and produce two minute wounds, from which only one or two drops of blood may at first issue—a smarting or severe burning pain is immediately perceived, the part begins to swell, and an œdematous puffiness, almost to the bursting of the skin, spreads in a few hours over the whole limb. The course of the lymphatic vessels becomes indicated by the red lines of inflammation: the glands swell. There is fever with delirium; small pulse, with pain in the region of the heart, and convulsions. These symptoms are attended with a feeling of anxiety and lassitude; laborious respiration; thirst, nausea, vomiting and syncope. Death from the bite of a viper has been known to occur in about thirty-six hours. If the individual survive the first effects, the wounded part may become livid and gangrenous; and he may sink under the irritative fever set up. According to Fontana, out of more that sixty cases, only two were fatal, and in one of these, gangrene commenced in the wound in three days, and the person died in twenty days. In one instance, a woman, æt. 64, died in thirty-seven hours, after having been bitten on the thigh by a viper. (Chelius's Surgery, Am. ed., vol. i. p. 389.) A fatal case occurred a few years since at St. Bartholomew's Hospital, in which the pain and swelling of the arm were followed by erysipelas and sloughing.

Such serious effects, however, are by no means common. A few years since I saw a girl who had been bitten in the lip by a caged viper, an hour after the occurrence, and the upper lip had swollen to nearly the size of the fist, producing great deformity. In this case there was some febrile irritation which disappeared in a few days.

With the exception of slight local and general irritation, it is rare to hear of any ill effects following the bite of the *common viper*. A case is reported by Mr. Taynton, in which a young man was bitten by a viper in the thumb of the right hand. The part began to swell in ten minutes. He was then seized with vomiting and purging. Half an hour after the accident, he was found with lividity of the skin, and the tongue was so swollen that there was difficulty in speaking. There was great prostration of strength, and the hand and arm became much swollen. Ammonia and opium were given; fomentations were applied to the arm; but the swelling did not subside for a week, and vesications formed around the wrist. (Med. Gaz. xii. 464.)

The bite of the **Rattle-snake**, (**Crotalus Horridus**, and C. **durissus**,) or of the **Cobra di Capello**, produces symptoms the same in kind, but much more severe and speedy in their progress. It may cause death almost immediately,—within a few hours (see Lancet, Sept. 21, 1839, 929,) or as a remote effect by leading to the sloughing of the cellular tissue of the limb. Small animals die in a few minutes. In an experiment on a small rabbit, the animal was bitten by a rattle-snake, it fell on its side, and died speedily in convulsions,—the abdomen becoming considerably distended before death. In man, nausea and great depression are among the early symptoms. The member bitten, and sometimes the whole of the body, begins to swell immediately after the wound is inflicted.

In a case in which a man was bitten twice in the hand by a **Rattle-snake**, (Crotalus Horridus,) the first symptom remarked was, incoherence of language and apparent intoxication. In half an hour the hand began to swell, then the fore-arm, and the pain extended to the axilla. In two hours the skin was cold, and the patient complained of sickness: syncope supervened. The day following there was considerable extravasation of blood beneath the skin. Vesications appeared on the wounded extremity, followed by abscesses, sloughing, diarrhœa, and delirium, in which state he died eighteen days after the bite. The only morbid appearance was turgidity of the vessels of the brain. (A very full account of this case will be found in Chelius's Surgery, Am. ed., vol. i. p. 393.) In the case of Mr. Drake who died in *nine hours* from the bite of a rattle-snake,—the membrane covering the brain and spinal cord had a reddish tinge. This formidable poison appears to act by completely destroying the vitality of the blood.

[See a Memoir on the poison of the C. **durissus** by Dr. R. Harlan. Trans. Am. Phil. Soc. N. S. III. 300 and 400.—G.]

Treatment.—The immediate application of a ligature between the part bitten and the heart, or of a cupping-glass, in order to prevent absorption. The wound should be enlarged and well washed. If absorption has taken place, and the limb is swollen, the whole of the skin may be smeared with oil, and attention must be directed to the constitutional symptoms. Brandy and ammonia may be given to prevent depression. Strong acetic acid which coagulates the poison, may be applied when the person is seen soon after the accident.

Nature of the poison.—The poison of the rattle-snake, according to Schlegel, (Physiognomy of Serpents, by Traill, p. 48,) is in its fresh state a transparent, limpid fluid of a greenish-yellow colour, slightly viscid, though less so than the saliva which it very much resembles. When dried it becomes viscid, and sticks firmly to substances,—it evaporates without inflaming; it is diffusible in water, but becomes opaline when shaken with it. It has neither an acid nor an alkaline reaction, and appears to be merely a mucous secretion; it has no odour, nor any taste. Russell applied one drop of the poison of the **Cobra di Capello** to his tongue and found it tasteless. It is commonly said that it may be swallowed with impunity, but Dr. Hering found while residing at Surinam, that on taking even small doses of it, much diluted with water, very perceptible effects were produced; such as pains throughout the body, abundant secretion of mucus from the nose and œsophagus, and diarrhœa. It is not certain that the poison which is said to have been swallowed with impunity, would have produced any effect had it been introduced into a wound. Dr. Meade describes the *viper-poison* as having a sharp burning taste. When a drop undiluted was placed on the tongue, the organ became swollen, and there was a sensation of soreness which remained for two days.

One remarkable circumstance is, that the poison retains its virulence even when dried. Some of the dried poison of the Cobra di Capello, which had the appearance of small fragments of gum arabic, was found by Dr. Christison to have retained its properties for fifteen years. A grain and a half dissolved in ten drops of water was introduced beneath the skin of a rabbit. In eight minutes the animal became feeble, and there were twitches of the body; it became torpid, there was great difficulty of breathing, and it died in twenty-seven minutes, without becoming insensible. The poison caused death by paralyzing the muscles of respiration. (On Poisons, 629.) It is probable that the virulence of the poison differs in different varieties, but nothing certain is known on this point. The Rattle-snake of America, and the Cobra di Capello of India, are the most formidable of the tribe. There is no known antidote to this poison. The serpent-charmers of the East secure themselves from injury by removing the poison-fangs, or by causing the snake to exhaust itself by biting other animals before proceeding to handle it.

POISON OF VENOMOUS CRUSTACEA AND INSECTS.

The Scorpion of the East and West Indies and Africa, has a poison-gland in the last joint of its tail, connected by means of a duct with the hollow sting. The poison is not fatal to large animals, but it produces severe pain, swelling, and erysipelatous inflammation of the limb. A lady at Kingston, in Jamaica, was stung by a scorpion in the foot, above the little toe. The part became instantly red and painful, and soon afterwards livid. The pain increased in severity, extending upwards, diffusing itself about the pit of the stomach, neck and throat, attended with depression, tremors, and cold perspiration. As the pain passed the abdomen, it occasioned violent purging and fainting, which ceased on its advancing higher. This lady did not recover for thirty-six hours. Some years since a man, stung by a scorpion, was admitted into the London Hospital. The hand and arm were much swollen and inflamed, and there was great nervous depression; but these symptoms soon subsided. (See Chelius's Surgery, Am. ed., vol. i. p. 387.) I have met with no case reported, in which the bite of the scorpion has proved fatal; but, according to Kirby and Spence, the black scorpion of Ceylon frequently inflicts a mortal wound.

The Scolopendra of South America and the West Indies, secretes a poison near the root of its strong horizontal pincers, which are tubular fangs. The wound inflicted by it is attended with the same effects as the sting of the scorpion. The scolopendra of Britain, being very small, can do no injury: but Dr. Traill speaks of one that came alive in a ship from Jamaica to Liverpool, which could extend its body to more than a foot in length, and could take considerable leaps.

[During a residence in one of the West Indian Islands, I heard of a case of death from the bite of a Scolopendra, and in this city a man lost an eye from a bite of one which came alive in a vessel from India.—G.]

The Spider secretes a poison which, when discharged through its fangs, kills flies and small insects. Some spiders in tropical climates are capable of inflicting severe wounds. The Tarantula of Southern Italy produces no injurious effects in the human subject by its bite.

The Argas Persicus is one of the Arachnidæ found only in the East. It has eight legs, and is furnished with a sucker. Its bite is poisonous, producing low fever.

The Bee, Humble Bee, the Wasp and Hornet, secrete a very acrid poison, producing the most intense pain and swelling of the part stung. Severe inflammation of the part has been known to follow, but this is rare. The poison-bag is in the tail, and is connected with the sting. I found, by causing a wasp to sting litmus paper, that the poison was acid. It is well known that oil or alkalies speedily relieve the pain, and this may be accounted for by the neutralization of the poison. The poison from one or a few of these insects, is in too small a quantity to do injury to man or the larger mammiferous animals; but instances are recorded where death has been produced by the stinging of a whole swarm. Dr. Gibson, however, mentions the case of a lady, æt. 69, who died in fifteen minutes after receiving the sting of a yellow wasp while engaged in drying apples; and one instance is reported, where a female lost her life by the sting of a bee in the œsophagus, which she swallowed in a piece of honeycomb. In his Lectures on Surgery, Mr. Lawrence describes the case of a gentleman who was stung about the throat and chest by a great number of bees, while he was endeavouring to raise a bee-hive which had fallen over. To those who ran to his assistance he said he felt himself sinking or dying; the action of the heart began to fail, the pulse sank, the breathing became interrupted, there was great anxiety, with nervous agitation and alarm, and he died in about ten minutes. (Chelius's Surgery, Am. ed., vol. i. p. 387.) There can be no doubt from the intense pain and irritation produced by the imponderable quantity of poison discharged through the minute sting, that it must be capable of producing formidable effects by concentration.

The common GNAT and the MOSQUITO (*Culex pipiens*,) are well known to produce irritating bites: they have no special sting, but they infuse a poison into the wound by their lancet-shaped suckers, which has the effect of accumulating the blood around the part bitten. The bites of other insects, like those of the HARVEST BUG (*Acarus autumnalis*,) are very irritating, but they are free from danger.

The reader will find some curious information on animal poisons in Traill's Outlines of Medical Jurisprudence, and Chelius's Surgery, loc. cit.

NARCOTIC POISONS.

CHAPTER XXXV.

GENERAL REMARKS ON NARCOTIC POISONS—OPIUM—PROPORTION OF MORPHIA CONTAINED IN IT—SOLUBILITY IN WATER AND ALCOHOL—LAUDANUM—SYMPTOMS—PERIOD OF COMMENCEMENT—THEIR RAPID AND SLOW ACCESSION—DEATH AFTER REMISSION—DIAGNOSIS OF POISONING BY OPIUM—CHRONIC POISONING—OPIUM-EATING—POST-PORTEM APPEARANCES—QUANTITY REQUIRED TO DESTROY LIFE—DEATH FROM SMALL, AND RECOVERY FROM LARGE DOSES—CONDITIONS WHICH MODIFY THE EFFECTS OF OPIUM—ITS ACTION ON INFANTS—EFFECTS OF DIVIDED DOSES—PERIOD AT WHICH DEATH TAKES PLACE—TREATMENT—RECOVERIES—POISONING BY POPPIES—GODFREY'S CORDIAL—DALBY'S CARMINATIVE—PAREGORIC ELIXIR—DOVER'S POWDER—OPIUM LOZENGES—MORPHIA AND ITS SALTS—EFFECTS OF ACETATE AND MURIATE—ABSORPTION—BLACK DROP—SEDATIVE SOLUTION—EFFECTS OF EXTERNAL APPLICATION—CODEIA—NARCOTINA—THEBAINA—NARCEIN—MECONIA—MECONIC ACID.

THE effects produced by the Narcotic class of poisons on the system have been already described (page 38, ante.) They are chiefly referable to disorder of the brain and nervous system. The most prominent symptoms, are headach, giddiness, paralysis, insensibility and convulsions. The brain is the organ, upon which a narcotic poison chiefly acts; but in some cases, by the occurrence of tetanus, there is an indication of a remote effect on the spinal marrow. The distinction between irritant and narcotic poisons is well-marked, so far as symptoms are concerned. Narcotic poisons are entirely destitute of any acrid or corrosive properties: they have no local chemical action on the mouth and fauces, and they very rarely give rise to vomiting or diarrhœa. When they prove fatal, they do not commonly leave any well-marked post-mortem appearances (p. 99, ante.) There is sometimes fulness of the cerebral vessels: but extravasation of blood is very rarely observed. It is usually said that they do not produce any redness of the mucous membrane of the stomach or intestines: this appearance has, however, been met with on several occasions in poisoning by Prussic acid. Opium does not cause inflammation of these organs, and when this condition has been found, it may probably be ascribed to the action of alcohol, in which the opium has been dissolved.

The Narcotics form a class of poisons so small that it is unnecessary to make any subdivision into groups. Toxicologists differ as to the number of poisons belonging to this class; but all agree in regarding Opium and Prussic acid as the most important in the series. The narcotic passes insensibly into the narcotico-irritant class, and hence there is great difficulty in assigning its exact limit, since poisons of the latter class in some instances act more violently on the brain, and in other cases on the spinal marrow. In the list of substances arranged among Narcotics (p. 41, ante,) I have been chiefly guided by the predominance of cerebral symptoms.

OPIUM.

General remarks.—OPIUM is a solid vegetable extract—the concrete juice of the unripe capsules of the *Papaver somniferum*. It is sometimes taken in this state as a poison, but more commonly in solution in alcohol under the form of tincture—LAUDANUM. Its poisonous properties are principally due to the presence of the alkali, *Morphia*, which exists in it in the state of a soluble salt, being combined with a particular acid, the *Meconic*. Opium contains a very variable proportion of morphia—the quantity varying from two per cent. in the Bengal variety, to about nine per cent. in certain varieties obtained from the East Indies. According to some chemists, good opium will yield from ten to thirteen per cent. of morphia. Dr. Ure states that it is difficult to procure more than seven per cent. M. Aubergier says that he has lately obtained from some varieties from 17·83 to 13·87 per cent. He thinks the proportion depends less on climate than on the species of poppy, and whether it be of the first, second, or third crop. (Gaz. Méd. Oct. 17, 1846, p. 824; see also Ann. d'Hyg. 1839, ii. 383.) The Turkey opium contains on an average about six per cent. according to the recent analyses of Mulder; but the best kinds of Smyrna opium contain thirteen per cent. (Brande, 1200.) This difference in the quantity of morphia contained in the drug, may sometimes account for certain differences observed in the effects produced by particular doses. It is said that the poisonous properties of morphia are much diminished by extraction from the drug: thus according to the per centage just given, about nine grains of opium are equivalent to half a grain of morphia: but it is well known that this dose of solid opium will produce a much more powerful effect than that quantity of morphia. Dr. Kelso, of Lisburn, doubts the correctness of this statement, from experiments made on himself. (Lancet, Sept. 1839.) Dr. Vassal asserts that the acetate of morphia has four times the strength of the aqueous extract of opium. According to Orfila, one part of morphia is equal in power to four parts of crude opium: but Dr. Christison thinks that half a grain is fully equal to two or perhaps three grains of the best Turkey opium. According to this view, it has six times the strength of opium.

There is no form of poisoning so frequent as that by opium and its various preparations. In two years, there were no less than one hundred and ninety-six fatal cases in England and Wales (p. 156,) forming nearly two-thirds of all the cases of poisoning that occur. One-seventh of these were cases of children poisoned by over-doses of opium or its compounds, and most of the others were the result of suicide or accident. It is calculated that *three-fourths* of all the deaths from opium, take place among children *under five years of age!* This, however, forms but a very small proportion of the actual number of cases; since there is no kind of poisoning wherein recoveries are so frequent. It is not often that we hear of a trial for murder by poisoning with opium:—the drug being seldom selected by murderers, except when the intended victim is a young child.

Solubility of opium in water.—The poisonous salt of opium, meconate of morphia, is soluble both in water, alcohol, and diluted acids. The aqueous and alcoholic solutions have an acid reaction. So far as I am aware, no experiments have been performed to determine the quantity of *opium* taken up by water in the form of infusion. In November 1843, a case of poisoning by opium was referred to me by Mr. T. O. Duke, of Kennington, in which this question arose. An ignorant nurse made an infusion by pouring hot water on powdered opium in a bottle, and gave, at short intervals, three teaspoonfuls of this infusion to a child aged about fourteen months, and it died, poisoned by the drug, in about eighteen hours. It was found that the infusion contained only 1·6 per cent. of solid matter, *i. e.* of the soluble part of the opium; and, that the principal part of the meconate of morphia had been taken up, was proved by an infusion which was subsequently made, retaining only very faint traces of this salt.

The following are the results of some experiments which were made in order to determine this question. Fifteen grains of finely-powdered opium were infused, for twenty hours, in six drachms of boiling distilled water. On examination, the filtered infusion was found to contain four per cent. of solid mater, *i. e.* of the soluble part of opium. In another experiment, opium sliced was employed with water in the same proportion. The quantity dissolved, averaged on several trials, from *three to four per cent.*, depending on the proportion of water and the length of contact. By boiling the residue in each case, a further quantity of meconate of morphia was obtained, showing that an aqueous infusion, while it will not extract the whole of the meconate at once, will certainly take up sufficient to render it actively poisonous to young children.

The *Extract of opium* is a medicinal preparation, and may be regarded as opium in a very pure form; it is obtained by macerating opium in a small quantity of water, and allowing the dregs to subside for some hours. Water dissolves the odorous principle, the salts of morphia and codeia, narcotina, gum, and other vegetable principles. Good opium is calculated to yield sixty per cent. of extract:—the average is from fifty to sixty. (See Ann. d'Hyg. 1839, ii. 384.) Three grains of the extract are, therefore, equal to about five grains of crude opium. It is employed in medicine in doses of from one quarter of a grain to three or four grains. A case of poisoning by this preparation will be related hereafter.

The poisonous salt of opium, meconate of morphia, is very readily dissolved by *alcohol.* This solution, which is of a dark colour, and possesses the powerful and peculiar odour of the drug, is commonly sold of a definite strength under the name of LAUDANUM, or TINCTURE OF OPIUM. The tincture of the London Pharmacopœia is said to contain about one grain of opium in nineteen drops; and from this datum, is commonly inferred the strength of those preparations of which it forms a part. I have, however, found by experiment, that one hundred grains of tincture of opium, carefully prepared according to the formula of the London Pharmacopœia, left on evaporation five grains of solid residue,—therefore it contained *one-twentieth* of the soluble portion of opium. Sixty minims of the same tincture weighed sixty grains, and left three grains of solid residue. The tincture is liable to vary in strength according to the goodness of the opium; but this variation is not observed to exist to any material extent. Dr. Pereira states that he has often procured morphia from the residue left after making the tincture (Op. cit. ii. 1773.) There is another fact, too, of some interest to those who prescribe the tincture. The morphia is easily precipitated by ammonia: thus, if the carbonate or aromatic spirits of ammonia be added to a mixture containing tincture of opium, the greater part of the morphia may be precipitated after twenty-four hours. Hence, if such a mixture is made a day or two before it is taken, the patient may by accident swallow the greater part of the morphia in the last remaining doses in the bottle. The presence of an excess of alcohol will, of course, prevent this precipitation.

When children are poisoned by opiate compounds, prescribed by ignorant persons, the medical opinion of the cause of death may be met by a very specious ex-post-facto defence; namely, that the particular tincture or preparation was not made according to the Pharmacopœia, but according to the druggist's own formula. It is very well known that fractional parts of a grain of opium cannot be easily separated from any opiate preparation; and that by no chemical process, can the exact quantity of opium in any mixture be determined. Hence this is a very convenient way of attempting to evade all kind of responsibility for the fatal result. Thus, if, by mistake or carelessness, one drachm of the tincture of opium be administered to a child and cause death with the usual symptoms of narcotism; and a medical witness proceed to infer from the known strength of the ordinary tincture, that at least three grains of opium were present in that dose, and were sufficient to destroy life, he may be met with the answer that the tincture in question was a private preparation, and not made according to the Pharmacopœia process. It need hardly be observed that if such a defence as this were admitted, no person could ever be convicted of poisoning under the circumstances. Perhaps the better rule would be for a witness to state from the symptoms and other facts whether there was or was not sufficient of the particular opiate preparation present to cause death, without speculating upon the precise fractional quantities of the drug to which this might correspond. The symptoms will commonly indicate whether they were or were not due to the medicine; and I fully believe that no judge or coroner, when there was a strong medical opinion in favour of poisoning, would permit an accused party to shelter himself under such an evasion as this. It would be equivalent to giving him the power of escaping all responsibility for mal-practice. The preparation used would be taken to be of the proper Pharmacopœial strength, unless the contrary were clearly made to appear. This kind of defence was set up in a well-marked instance of poisoning by paragoric, in which Dr. Babington, Mr. M'Cann, and myself, were consulted (G. H. Reports, April 1844;) although there could be no medical doubt that death was caused by the poison, and a verdict was returned accordingly.

The rate at which this poison (laudanum) is retailed to the public, is as follows,—from half a drachm to two drachms for twopence, from two drachms to four drachms for fourpence,—exceeding this quantity, eightpence and one shilling per ounce. It is very often sold by ignorant drug-dealers for Tincture of Rhubarb.

[The prices in Philadelphia for Laudanum vary exceedingly, as does also the quality of the Tincture: for the article of proper strength, our druggists charge about three cents for two drachms, six cents for half an ounce and twelve cents for an ounce.—G.]

SYMPTOMS.

The symptoms which manifest themselves when a large dose of opium or its tincture has been taken, are of a very uniform character. They consist in giddiness, drowsiness, and stupor, succeeded by perfect insensibility, the person lying motionless, with the eyes closed as if in a sound sleep. In this stage he may be easily roused by a loud noise, and made to answer a question; but he speedily relapses into stupor. In a later stage, when coma has supervened with stertorous breathing, it will be difficult, if not impossible, to rouse him. The pulse is at first small, quick, and irregular, the respiration hurried, and the skin warmed and bathed in perspiration—sometimes livid; but when the individual becomes comatose, the breathing is slow and stertorous: the pulse slow and full. The skin is cold and pallid. The pupils are sometimes contracted, at others dilated. From cases which I have been able to collect, contraction of the pupils is much more frequent than dilatation. In a case referred to me in 1846, one pupil was contracted and the other dilated. They are commonly insensible to light. The expression of the countenance is placid, pale, and ghastly: the lips are livid. Sometimes there is vomiting, or even diarrhœa; if vomiting take place freely before stupor sets in, there is great hope of recovery. This symptom is chiefly observed when a large dose of opium has been taken; and it may be perhaps ascribed to a mechanical effect of the poison on the stomach. Nausea and vomiting, with headach, loss of appetite, and lassitude, often follow on recovery. The muscles of the limbs feel flabby and relaxed, the lower jaw drops, the pulse becomes feeble and imperceptible, the sphincters are in a state of relaxation, the temperature of the body is low, there is a loud mucous rattle in breathing, and convulsions are sometimes observed before death: these are more commonly met with in young children than in adults. One of the marked effects of this poison is to suspend all the secretions except that of the skin. During the lethargic state, the skin, although cold, is often copiously bathed in perspiration. It is a question yet to be determined, whether this may not be the medium by which the poison is principally eliminated.

These symptoms usually *commence* in from *half an hour* to *an hour* after the poison has been swallowed. Sometimes they come on in a few minutes, especially in young children; and at others their appearance is protracted for a long period. In a case reported by Dr. Skae, the individual was found totally insensible in *fifteen minutes*. As we might expect, from the facts connected with the absorption of poisons, when the drug is taken in the *solid* state, the symptoms are commonly more slow in appearing, than when it is *dissolved* in alcohol. Their appearance is also protracted if the stomach be full at the time: and it is said that intoxication has the effect of retarding them. In a case reported by Mr. Semple (May 1841,) a dose of one ounce and a half of laudanum was taken by a girl, aged nineteen:—the symptoms did not appear until *an hour and a half* afterwards. There was drowsiness, but the patient was rational, and the pupils were contracted to the size of a pin's head, and did not dilate on removing the light. Under treatment the girl recovered. With the exception of an extraordinary instance mentioned by Dr. Christison, the longest period to which the symptoms have been protracted was *five hours*. This occurred in a case reported by Dr. Sewell. A man, æt. 40, swallowed ten drachms of tincture of opium by mistake for tincture of rhubarb. When seen by Dr. Sewell, five hours afterwards, he was in bed, awake, and quite conscious. The pupils were contracted to the size of a pin's point, and immoveable; the temporal arteries pulsated with great violence; the speech was uttered with difficulty, the skin dry, pulse 100 and jerking. By active treatment, the man recovered in about fifteen hours. (Dublin Hosp. Gaz., Oct. 15, 1845, p. 78.) In a case in which I was consulted by Mr. Mann, of Boxford, a young female died in about twelve hours from the effects of a large dose of opium, and there was some reason to believe that the confirmed symptoms of narcotic poisoning did not show themselves until *three hours* after she had swallowed the poison. (Med. Gaz. vol. xxxvii. 724.) In Dr. Christison's case, a man swallowed an ounce and a half of laudanum, and in an hour, half as much more, and lay down in bed. Some excitement followed, with numbness of the extremities. He continued sensible, and so lively for *seven* hours after the first dose, that a medical man did not believe his statement. It was not until the *eighteenth* hour that stupor set in, and in two hours more the symptoms of poisoning by opium were of an aggravated kind. He finally recovered under treatment, but the cause of this extreme protraction of the symptoms could not be ascertained. (Op. cit. 706.)

The period at which the cerebral symptoms commence, is a question of some importance in relation to the retention of power on the part of the deceased to perform certain acts indicative of volition and locomotion after having swallowed a large dose of this poison. Thus the narcotic effects may not come on, until the deceased has had ample time to attempt suicide in some other way. In March 1843, a gentleman committed suicide at Hammersmith: he was found suspended by a silk handkerchief; but it was shown that he had previously swallowed a large dose of laudanum. There was no doubt that he had died from hanging. In general, it must be allowed as at least possible, that a person who has taken a sufficient quantity of this poison to prove fatal, may move about and perform many acts for one or two hours afterwards, but this power ceases when the symptoms are confirmed. (See case by Dr. Skae, p. 480.)

Death after remission of the symptoms.—It has been frequently observed, in cases of poisoning by this drug, that the individual has recovered from the first symptoms, and has then had a relapse and died. There is some medicolegal interest connected with this state, which has been called secondary asphyxia from opium, although there appears to be no good reason for giving to it this name. In December 1843, a gentleman swallowed a quantity of laudanum, and was found labouring under the usual symptoms. The greater part of the poison was removed from the stomach by the pump; and he so far recovered from his insensibility, as to be able to enter into conversation with his medical attendant; but a relapse took place, and he died the following night. It is not improbable that, in these cases, death may be occasioned by the accumulation of the poison, carried by the absorbents into the system; *i. e.* the morphia may be more rapidly carried into the system than it is eliminated out of it. A very remarkable case illustrative of this *remittent* form of poisoning by opium has been published by Mr. Kirby. (Dubl. Med. Press, Dec. 24, 1845, 406.) Mrs. J., aged thirty, a lady of slender form, hypochondriacal, and very irritable, was in the habit of taking frequently, thirty drops of laudanum for the purpose of relieving certain distressing sensations. On the 27th of February, in a moment of passion, she swallowed six dessert spoonfuls, about *an ounce* of the common tincture of opium, having previously taken four glasses of port wine and some spirits in her tea. After three hours she was found in her bed, and perfectly insensible. She was then deadly pale and cold; her limbs were relaxed, and felt flabby when handled. She breathed slowly, and apparently with great difficulty. There was a loud mucous rattle in the trachea, with great frothing at the mouth, which was wide open, the under jaw having fallen towards the sternum. The eyelids were closed, and when raised by the finger, exhibited a distorted, fixed, suffused eyeball, with an extremely contracted pupil; pulse 100, and exceedingly feeble; free from intermission, although the motions of the heart were tumultuous. The temperature of the body was generally very low, but it was high at the præcordium. There was paralysis of the muscles of deglutition, with intense coma. Fourteen hours were fruitlessly employed in the administration of emetics, stimulating enemata of the sulphate of zinc, and frictions. Towards morning she could swallow small quantities of tea, coffee, broth, bread, and ammonia at intervals, and complained only of her great distress in breathing. Soon afterwards she recognised her medical attendant, and knew all her domestics. She detailed all the proceedings of the preceding night, as they have been given, regretted what she had done, and expressed much anxiety as to the hapless and hopeless result. The limbs were still powerless; the eyelids were raised, but only by a great effort, and they quickly resumed a half-open position, exposing suffused balls, a persisting contraction of the pupil, and a frightful strabismus. For a moment she appeared able to direct them to an object. They then rolled as if free from control, and settled into quiescence, the cornea being turned upwards and wholly concealed. On a few occasions she vomited a small quantity of fluid darker than coffee, and without any odour of opium. She partook of strong chicken-broth, and was soon capable of conversing with a minister, from whose hands she received the sacrament.

This excitement over, being about eighteen hours from the time of the fatal dose, the difficulty of breathing became an agony. She referred all her sufferings to her "lungs," asked for a blister to her back, and announced that she was surely dying. The wrist was now pulseless; the extremities were cold; the limbs were relaxed, and she died in twenty-three hours after she swallowed the laudanum. She retained her consciousness until a few moments before she expired. The body was inspected nineteen hours after death: it was then warm and very rigid. The extremities were strongly extended, and the hands clenched. The whole front was pale, while all the underlying parts were deeply ecchymosed. The lungs filled the chest, and scarcely retired on exposure; they crepitated when handled, and yet they felt firm and fleshy, and resisted the scalpel. Their anterior surface presented an anæmic appearance, studded with small black spots of a circular form. Internally, they were gorged with livid blood. The large pulmonary vessels were nearly empty, as was the heart, the blood which they contained being fluid. The liquor pericardii amounted to half an ounce. The stomach was distended by a brownish-coloured inodorous fluid. The internal tunic exhibited purple marks, more attributable to vascular turgescence than to extravasation. The intestines were pale throughout, except near the gall-bladder, where they were extremely and deeply tinged with bile, and they were undistended by flatus. The voluntary muscles were peculiarly florid. Examination of the head was not permitted.

This power of recovery, followed by death in a few hours, might become a serious question in a medico-judicial inquiry. It will be observed that in this instance, it extended only to consciousness and sense. There was no restoration of the power of locomotion. The muscular system appears to have been in a state of complete paralysis. Dr. Christison quotes a case from Pyl in which, after a large dose of opium, there was a complete remission of symptoms; but paralysis came on, and the man died in ten days. (Op. cit. 710.) He thinks that opium could only act in this manner by calling forth some disposition to natural disease—some organic affection of the brain being suddenly developed through the cerebral congestion occasioned by the poison. In reference to Pyl's protracted case this may be the true explanation; but in other instances cited, we can only, it appears to me, refer the fatal symptoms, notwithstanding the remission, to the direct effect of the poison. After all, at an inquest the question would be reduced to this:—Would the deceased have died under the same circumstances had he not taken the dose of opium? Whether the drug acted directly to destroy life, or indirectly by inducing some fatal disease, it must equally be regarded as the immediate cause of death,

Diagnosis of poisoning by opium.—Cases sometimes present themselves in which individuals die under symptoms closely resembling narcotic poisoning, and should any opium have been given by the medical attendant, a serious question of malapraxis is likely to arise. In severe *burns and scalds*, the brain often participates in the injury, and stupor frequently precedes death. In a case related by Mr. Abernethy, a medical man was charged with the manslaughter of a child by giving to it an over-dose of opium, while it was labouring under the effects of a severe scald. Mr. Abernethy stated in his evidence, which was given in favour of the practitioner, that he thought the exhibition of opium in such a case very proper:—that the quantity given, eight drops of tincture of opium immediately after the accident, and ten drops two hours afterwards, was not an over-dose for a child: the circumstance of the child continuing to sleep until it died, after the exhibition of the opium, was no proof that it had been poisoned. This sleep was nothing more than the torpor into which it had been plunged by the accident. The surgeon, upon this evidence, was acquitted. It is very likely that Mr. Abernethy may have taken a correct view of this case; but as the *age* of the child is not stated, it is impossible to form an opinion respecting the adequacy of the dose prescribed, to occasion death. We shall see hereafter that young children are very easily destroyed by doses much smaller than those administered in this instance. An investigation of great interest in this point of view took place at Putney, in July 1847. Dr. Wane, who was entrusted with the post-mortem examination, communicated to me the following particulars:—A young lady, æt. about 14, died suddenly soon after she had taken some opiate medicine, and had applied tinctura of opium as an embrocation. When the medical attendant was summoned, he found his patient insensible, and as he thought, in a state of narcotism from the medicine. The deceased, according to one account, had taken three pills, consisting of *half a grain* of opium and half a grain of extract of hyoscyamus (one to be taken every hour,) and the liniment was composed of oil of turpentine and liniment of the susquicarbonate of ammonia, each five drachms, tincture of opium two drachms: the greater part of this was rubbed on the skin of the abdomen. Besides, it was stated in evidence that the patient took in two doses, a mixture containing a solution of muriate of morphia equivalent to about half a grain of opium. There is every reason to believe that the deceased did not take on the whole, more than *a grain* of opium, and there was nothing in the disease under which she was labouring, to aggravate its effects. It was about eight hours after the medicines had been taken that she was found insensible, with a scarcely perceptible pulse, and she soon afterwards died. The medical attendant who prescribed the medicine, suspected at the time that opium might have been the cause of the symptoms, and at his application an inquest was held. The appearances found on inspection were great congestion of the vessels of the pia mater, as well as of the substance of the brain, with effusion under the tunica arachnoides; in the abdomen there were all the marks of *enteritis* in its most intense form, and to this death was properly ascribed, the apparent narcotism having been nothing more than the stupor which often precedes death in such cases. The quantity of opium taken by the deceased, was certainly insufficient to account for the symptoms and fatal result. (See Month. Jour. Med. Science, Aug. 1847, p. 131.)

It is very well known that coma and convulsions are among the symptoms which precede death under that form of death dependent on *granular degeneration of the kidney*. This is ascribed to the poisoning of the blood by urea. The fact is important, inasmuch as a suspicion might easily arise in a fatal case, where an ordinary dose of opium had been given in the course of treatment, that death had been caused by the drug. Mr. Moore has communicated an interesting case involving this question, to the Medical Gazette. (xxxvi. 821.) A man, æt. 39, was admitted into the Queen's Hospital, Birmingham, labouring under the following symptoms:—breathing laborious, at times stertorous; stupor; on being roused he answered questions readily, but with a stolid aspect; the muscles flaccid; pupils of moderate size, answering perfectly to light; conjunctivæ pallid; pulse 94, full, but weak; occasional convulsive twichings of the upper extremities. It appeared that the man, having suffered for a few days from diarrhœa, went to a druggist's and asked for some tincture of rhubarb. A large quantity was poured out of one bottle into which a few drops of another liquid were dropped. The man drank the dose in the shop: he soon afterwards became drowsy, returned to the druggist's, and asked whether laudanum had been given him by mistake. He was informed that a few drops had been put into the rhubarb on account of the severity of the symptoms. On reaching home he fell asleep, and continued sleeping, except when temporarily roused, until his admission. He vomited shortly after taking the draught, but the matter ejected had been thrown away. The stimulating and ambulatory treatment adopted in narcotic poisoning, was resorted to with only a temporary removal of the somnolency. The man still relapsed into a lethargic state, and even galvanism was employed with little benefit. The breathing became more oppressed; the extremities were cold and clammy, and the man was with great difficulty roused; the pulse was 60, and feeble: pupils slightly contractile, and the stertor increased. Bleeding was attempted, but it had an injurious effect. The man died forty-four hours after his admission, and one hundred and two hours from the time of taking the dose of rhubarb and laudanum. The body was inspected twenty-one hours after death. The blood-vessels of the membranes of the brain were slightly congested posteriorly; but the brain itself was firm, pale, and exsanguine. The lungs were much congested and infiltrated with serum. The stomach contained two ounces of a yellow pultaceous matter having no smell of opium, and yielding no traces of this drug on analysis. The mucous membrane was sound but congested; that of the intestines, with general congestion, presented several spots of ulceration. Both of the kidneys were in a state of granular degeneration: the urine contained in the bladder was albuminous, and urea was obtained by analysis, from the brain..

It will be observed that this case closely resembled one of poisoning by opium, in the time of the occurrence, and in the character and progress of the symptoms. The occurrence of a remission, as it has been already stated, is not against this view (page 470, ante.) It differed from the effects of opium in the state of the pupils, which do not appear to have become contracted, except towards the last; and in the condition of the brain, which was not congested. At the inquest, the druggist's assistant deposed that he had given the deceased four drachms of the tincture of rhubarb, into which he had dropped only *five drops* of laudanum. It is not at all probable that this small quantity (equal to one quarter of a grain) would have occasioned the intense somnolency and death after so long a period in an adult, but it might be assumed from the similar appearance of the tinctures, that the one had been accidentally substituted for the other; and thus four drachms of laudanum (equal to *twelve grains* of opium) had in reality been given to the deceased. On this assumption the whole of the facts, excepting the state of the pupils and brain, which might be regarded as anomalous conditions, would admit of an easy explanation. Unfortunately, the only method by which this mistake could have been detected was here inapplicable, the matter vomited by the deceased soon after he had taken the draught, having been thrown away! He did not come to the hospital until fifty-two hours after he had taken it; and as he survived between four and five days, and vomiting appeared among the early symptoms, it is not at all probable that any traces of half an ounce of the tincture of opium would be found in the stomach after so long a period had elapsed. Mr. Moore deposed at the inquest that the immediate cause of death was congestion of the lungs, which might have been produced by a poisonous dose of laudanum, but that the disease of the kidneys, which was present, would fully account for it, and that this latter cause was rendered more probable by the discovery of urea in the brain.

For the reason mentioned, the real cause of death was obscure. It is quite possible that a person labouring under granular degeneration of the kidneys, with albuminous urine, and in whose brain urea may be detected, may die from half an ounce of laudanum; and there is nothing in the case to show that such a mistake, by no means unusual in the preparation of draughts extemporaneously, could not have been made in this instance.

One of the great distinctions between the *coma* of disease and that of poisoning by opium, is that in the latter only, can the individual be readily roused and made to have a temporary consciousness. In a doubtful case, therefore, this condition should be looked for. It would be proper in all suspicious instances, where coma, with or without convulsions, precedes death, to examine the state of the kidneys—the quantity of opium given may be ascertainable, and this granular condition of the kidneys may then materially aid the diagnosis. The urine may, however, be albuminous, independently of the existence of this granular degeneration; and Dr. Rees has justly observed that urea will be found in the blood in undue proportion in various conditions of the body, characterized by ischuria to any extent. (Anal. of Blood and Urine, 2d edit., 159.) For many other facts connected with the diagnosis of narcotic poisoning from various other diseases, see ante, page 58.

The diagnosis of these cases is not merely important in a medico-legal view, but it is essential in order to direct the treatment. Thus the means required in a case of narcotic poisoning, would only accelerate death if the symptoms were owing to disease. In one instance, sulphate of zinc and brandy, with forced motion in walking, were resorted to, when the inspection showed that death had proceeded from disease, and not from opium!

Chronic poisoning by opium.—Opium-eating.—When opium is taken for a long period in small doses which are gradually increased, its effects are very different. It is this state which we witness in those individuals who are addicted to opium-eating. The injurious effects of the drug thus taken for a long period of time, have already given rise to an important question in law relative to life-insurance; and it will be, therefore, proper to state those facts which have been ascertained with respect to the influence of this practice on health. One of the best descriptions of the effects of opium-eating is that given by Dr. Oppenheim in his account of the state of medicine in Turkey. He says, "The habitual opium-eater is readily recognised by his appearance. A total attenuation of body,—a withered yellow countenance,—a lame gait,—a bending of the spine, frequently to such a degree as to cause the body to assume a circular form,—and glassy deep-sunken eyes,—betray him at the first glance. The digestive organs are in the highest degree disturbed; the sufferer eats scarcely any thing, and has hardly one evacuation in a week; his mental and bodily powers are destroyed. As the habit becomes more confirmed, his strength continues decreasing, the craving for the stimulus becomes greater; and, in order to produce the desired effect, the dose must be constantly augmented. After long indulgence, the opium-eater becomes subject to neuralgic pains, to which opium itself brings no relief. These persons seldom attain the age of forty if they have begun to use opium early." This description of the effects, is exactly what we should expect from physiological and pathological reasoning. Dr. Christison states, that he has ascertained that constipation is by no means a general consequence of the continued use of opium; but this may be an exception to the rule. It is believed by some that the action of the drug may be different in different countries, and that the description of the effects produced by the use of opium in Turkey, cannot be applied to the English opium-eater. The following case, however, which occurred to Dr. A. T. Thomson at University College Hospital, is adverse to this view:—

E. M., aged 35, was admitted May 26th, 1835. About seventeen years before she began to suffer from a pain in the right iliac region, for which a medical gentleman ordered her to take *ten drops* of laudanum, night and morning. This was gradually increased, the pain continuing, until at last she took three teaspoonfuls every four hours, night and day. At first the ten drops relieved the pain; but it was found necessary to increase the dose to produce the same effect; so that the three teaspoonfuls at last did not produce so much relief as did the ten drops at first. The effect of the small doses was simply to produce a relief from the pain, without exerting any other particular influence on the body or mind. As the dose was increased, however, she found that it produced a very comfortable condition of the mind. She felt lively and cheerful, and was capable of doing any amount of work; it also produced a sense of warmth over the whole body. She had severe family afflictions, but was not at all distressed by them whilst under the influence of opium, though she felt them severely at other times. If she passed over the usual time for taking a dose, she felt the most distressing sensations about the joints, not of pain, but such as she was unable to describe. She suffered from involuntary motions of the arms, fingers and toes; numbness in the limbs and body generally; profuse perspiration, nausea, vomiting, and loss of appetite; a saline taste in the saliva, and a bad taste in the mouth; tremor in the limbs; great sense of debility and lassitude. The memory and mental powers generally became greatly impaired, attended by a miserable depression of the spirits. These symptoms are all relieved by a repetition of the dose. The opium also produced constipation,—not more than one motion occurring in a week; and she does not recollect whether this was produced by medicine or not. If the dose was deferred, she always suffered from severe headach. Her sense of smell was so much impaired that she could perceive no pungency in snuff; her taste was so much lost that she could not distinguish pepper or mustard; and her hearing had become so defective that she could hardly detect the voice of any one who spoke; yet her own voice sounded most disagreeably loud to her. Her touch was so much affected that she could not execute any needlework. The acuteness of all her senses was restored by the usual dose, the want of which was indicated by flushing and heat of the face. During the period of taking the opium, she had very little sleep, and in the intervals she could not attempt to sleep, from want of the desire, so that she generally worked all night. What sleep she had was generally in the daytime, and that little was much confused, and easily ended. About five or six years ago, her resources being exhausted, she obtained admission into the hospital. Her laudanum here was left off for the first three days, and all the above symptoms continued; she now, for the first time, appeared to see the most frightful animals and other objects in the ward. The symptoms were again relieved by her usual doses. The laudanum was decreased during the whole time; and when she left the hospital she took only a teaspoonful in the course of the day. On returning home, and being dependent on her friends, she was obliged to discontinue the laudanum and wine, and was even unable to get beer. She was now more miserable than before, all the symptoms returning with increased severity; and for the first six months she was almost entirely helpless. She was then first affected with pain in her chest and a cough, which has continued ever since. She was twelve months at home before the above distressing symptoms disappeared. The only consequences of her opium-eating at present, are a much impaired taste, numbness of the limbs, coldness of the feet, inability to walk far without aching pains in the limbs, and a general sense of lassitude.

There is also abundant evidence that this drug, as it has been administered to children in the Factory districts, has produced serious detriment to health. In Mr. Grainger's Report on the Children's Employment Commission, it is stated that laudanum and other preparations of opium are given to young children in gradually increased doses, until the child will bear from fifteen to twenty drops of laudanum at a time. The child becomes pale and wan, with a peculiar sharpness of feature, and rapidly wastes away. The majority of these children die by the time they are two years old, (ante, p. 36.) These facts appear to show that climate does not at all affect the action of the drug in the early periods of life; and I do not think that the observations yet made, are sufficiently numerous to justify us in affirming the existence of this influence with respect to adults. It has been truly remarked that many may die young from the effects of opium-eating, without the secret being discovered; for even the medical attendant may be kept in complete ignorance of his patient indulging in it.

In 1826, the Earl of Mar effected an insurance on his life; and two years afterwards, *i. e.* in 1828, he died of jaundice and dropsy, at the age of fifty-seven. The Insurance company declined paying the amount of the policy, on the ground that the earl was, at the time of the assurance, and had been for some time previously, an opium eater. This practice was concealed from the insurers; and it was further alleged that it had a tendency to shorten life. It was clearly proved in evidence that the earl had been a confirmed opium-eater up to the time of his death. According to Dr. Christison, he used laudanum for thirty years, at times to the amount of two or three ounces daily, taking a table-spoonful for a dose. He was a martyr to rheumatism, and, besides, lived rather freely. Many individuals who were constantly about him, and many intimate friends, deposed, that until 1826 (the year of the insurance) he was of a cheerful disposition, and clear in his intellects. Some of them admitted that they then perceived a change in his habits, which they attributed to the adverse circumstances in which he was compelled to live. In 1825 Dr. Abercrombie found him enfeebled, and broken down in constitution, but without any definite complaint. The main question at the trial was, whether opium-eating had a tendency to shorten life; for on this turned the issue,—whether the concealment from, or the non-communication of the practice to, the Office, was or was not material.

Drs. Christison, Alison, Abercrombie, and Duncan, were examined on the part of the Insurance company; and, although they entertained the opinion that the habit had a tendency to shorten life, they could not adduce any cases in support of it. This opinion was based on the general effects of opium, as manifested by its action on the brain,—by its producing disorder of the digestive organs, and giving the individual a worn and emaciated appearance. In most of the instances collected, there was no evidence that life had been shortened by the practice. On the contrary, some of the individuals had carried it on for years, and had attained a good old age. The jury returned a verdict for the plaintiffs, not on the ground that the practice was innoxious, and its concealment immaterial, so much as on the technical point that the insurers had not made the usual and careful inquiries into the habits of the deceased; and they were therefore considered as having taken upon themselves the risk of their own *lâches*. It appears that the *general* question with respect to habits, was not answered by the medical referee; and it was therefore assumed that the Office had taken the risk and waived the knowledge of them. A new trial was granted, on the ground of misdirection; but the suit was compromised.

Hence it will be seen no decision was come to on this important question, which is very likely to arise again. It is therefore proper to examine some of the facts connected with opium-eating, in order, if possible, to see how far it really tends to shorten life. In the case of the Earl of Mar it appeared to be a fair inference that the habit did not shorten life; for he is represented to have indulged in it for thirty years; and for twenty-eight years, according to the statements of his friends, no injurious effects had followed. Dr. Christison has collected no less than twenty-five cases from numerous quarters, by which we learn that opium has been taken in large quantities for forty years together, without producing any marked injury to health. At a meeting of the London Medical Society, Dr. Clutterbuck related the case of a woman who for seven years had taken two scruples of solid opium daily. She was fifty-four, had led an irregular life, and had first taken opium to relieve the pains of rheumatism. The dose was not increased, and the usual ill effects of opium were absent,—such as constipation, nausea, and loss of appetite. Although she did not increase the dose, the effects of the diminution of a single grain of her usual quantity were most marked, and she immediately perceived them. Many instances of a similar kind are recorded.* Such cases appear to show that opium-eating has not necessarily that tendency to shorten life which it has been hitherto supposed to have. There is, however, sufficient evidence to prove that the practice gives rise to prejudicial effects on the system, and tends to impair health. It may not have this effect in all cases, except on the withdrawal of the stimulus; but this is not the question. It might be on this principle argued that the drinking of alcoholic liquids has no tendency to shorten life, because some hundreds of cases may be adduced in which the individuals have been addicted to intemperate habits for years, and have still appeared to suffer but little in bodily health. All who have witnessed the effects of opium-eating in Turkey and China, agree that the practice leads to the speedy destruction of health, and it is therefore calculated to shorten life. In a case of proposed life-insurance, the insurers ought to be informed of this habit, where it exists; and no medical man should sanction its concealment merely because many addicted to it, have lived for years in apparently tolerable health. One of the questions put to a medical man is, whether he knows of any material circumstance touching the health or habits of the person to which the other questions in the certificate do not extend; and, if so, he is required to state them. Now, without going the length of saying that the life of an opium-eater is uninsurable upon a common risk, the habit is itself sufficiently material to require that it should be declared in reply to such a question as this. The practice may be, and often is, concealed from a medical attendant; then the assured, if not candid in avowing its existence, must expose his representatives to the risk of losing all benefit under the policy. Independently of medical data, which appear to favour both sides of this question, the jury would probably be guided by the effect actually produced on the constitution of an individual who had been addicted to the practice. If it have continued for many years, and there were no proof of his health having in consequence undergone any remarkable change, this might be regarded by the jury as the best possible evidence in favour of the concealment not being in that case material. The insurers could not equitably complain of the verdict in the Earl of Mar's case; for, as he began opium-eating at twenty-seven, and died at fifty-seven, without any obvious injurious effects being produced by the use of the drug, it could not be said that, in his case at least, the practice had shortened life. I do not know that it is in our power to apply any better or more practical test than this, under circumstances in which medical facts appear to bear both ways. The habit is very different from the abuse of alcoholic liquids. No one can doubt that in this form of intemperance, the result must be inevitably to impair health and to shorten life. The facts here bear only one way; and, if instances of longevity can be adduced among spirit-drinkers, they are well known and generally admitted to be exceptions to the rule. The queries put by Insurance offices are now so explicit, that they must be considered as including the habit of opium-eating; and there does not appear to be any just pretence for evading the admission of the practice, either on the part of the insured or (if known to him) of his medical attendant.

POST-MORTEM APPEARANCES.

In a case which proved fatal in fifteen hours, examined at Guy's Hospital, a few years since, the vessels of the head were found unusually turgid throughout. On the surface of the anterior part of the left hemisphere there was ecchymosis, apparently produced by the effusion of a few drops of blood. There were numerous bloody points on the cut surface of the brain:—there was no serum collected in the ventricles. The stomach was quite healthy. Fluidity of the blood is mentioned as a common appearance in cases of poisoning by opium. There is also engorgement of the lungs; most frequently according to Dr. Christison, in those cases which have been preceded by convulsions. (Op. cit. 732.) Among the external appearances there is often great lividity of the skin. This may be taken as a fair statement of the post-mortem appearances in poisoning by opium. Extravasation of blood on the brain is rarely seen;—serous effusion in the ventricles, or between the membranes is much more common. The stomach is so seldom found otherwise than in a healthy state, that the inflammatory redness said to have been occasionally met with, may be regarded as probably due to accidental causes. When tincture of opium has been taken and retained on the stomach, increased vascularity in the mucous membrane may be occasionally produced by the alcohol alone.

A case was referred to me by Mr. Colgate, in August 1844, in which a woman, æt. 56, died in twenty-two hours after taking *half an ounce* of tincture of opium by mistake for tincture of rhubarb. The body was inspected twenty-four hours after death, and the following appearances were met with. The brain was congested throughout, the blood in the sinuses fluid, and there was about an ounce of serum effused between the membranes and in the ventricles. The heart presented a cartilaginous hardness near the tricuspid valves, but not so as to interfere with their action. The lungs were emphysematous in places, and there were some small tubercles scattered over them, chiefly in the right lung. The liver was rather enlarged at the superior part of the right lobe, and considerably congested at the lower extremity. The intestines, nearly empty, had patches of congestion, with bloody mucus on the lining membrane. The spleen was rather large:—the rest of the body quite healthy. The stomach had become partially decomposed:—at the larger end there was some redness of the mucous membrane, but apparently of a pseudo-morbid character. The contents consisted of a dark, pasty, offensive mass, of a greenish-colour, intermixed with lumps of half-digested animal food: there was no smell of opium to be perceived, nor could the least trace of meconic acid or morphia be detected in them by the most careful analysis.

In a case of poisoning by a large dose of tincture of opium, Dr. Sharkey found the following appearances twelve hours after death. The body warm and rigid; the stomach healthy, containing a quantity of a gruel-like fluid, without any smell of opium. Intestinal canal and all the other viscera healthy. The veins of the scalp, as well as of the dura mater and sinuses, were gorged with blood; but there was no effusion in any part of the brain. The contents of the stomach yielded no trace of morphia or meconic acid, but there was no doubt that death had been caused by opium, taken the previous night. (Med. Gáz. xxxvii. 235.)

This description of the appearances refers to the action of large doses on adults. In a case which I had to investigate a few years since, a child aged fourteeen months was killed in eighteen hours, from the effects of a dose of infusion of opium equivalent to from three to five grains of the powder. The inspection of the body was made about twenty-four hours after death. It was not emaciated; and, externally, there were no particular appearances, excepting a few livid spots on the skin of the abdomen, back, and genitals, as also on the upper part of the thighs, sides, and back of the neck. The eyelids were open; the eyes sunk into the orbits, and their transparency gone: the child it seems, had died with its eyes prominent and open. The pupils appeared contracted, but the condition of the iris was not particularly noticed during life. The viscera of the chest were perfectly healthy; there was no mark of effusion, or of any organic disease. The right cavities of the heart were congested, and the lining membrane of the organ was observed to be somewhat opalescent. The viscera of the abdomen were also healthy, except the kidneys, the cortical structure of which had undergone some change from disease, although to a very slight extent. It had evidently had no influence on the illness and death of the child. The peritoneum presented, in some parts, patches of a milky whiteness; but there was no appearance of inflammation or effusion. The stomach was perfectly healthy; the mucous membrane was raised into numerous rugæ, but there was no trace of inflammation or disease in any part. The cavity of the organ contained about a teaspoonful of a white viscid liquid, apparently consisting of milk and mucus in a semi-digested state. There was no farinaceous or any other food present, and no smell of opium; nor was the slightest trace of morphia or meconic acid detected in it on analysis, although the child had not vomited, but had remained throughout in a state of insensibility. The intestines were found quite healthy. On opening the duodenum and jejunum a small quantity of liquid, similar to that contained in the stomach, was observed: this was also collected and set aside for analysis. In the cranium, the blood-vessels of the brain were found much congested; but there was no effusion or extravasation of blood or serum. In all other respects the brain presented its usually healthy characters.

From this account of the post-mortem appearances it will be seen that there is nothing but turgescence of the vessels of the brain, which can be looked upon as indicative of poisoning by opium, and even this is not always present. This condition of the brain, however, if it exist, can furnish no evidence of poisoning, when taken alone, since it is so frequently found, as a result of morbid causes, in otherwise healthy subjects.

QUANTITY REQUIRED TO DESTROY LIFE.

The medicinal *dose* of opium, in *extract* or *powder*, for a healthy adult, varies from half a grain to two grains. Five grains would be a very full dose. The medical dose of the *tincture* is from ten drops to one drachm,—as an average from *thirty* to *forty drops*. Very large doses may be borne in cases of hydrophobia, colic, delirium tremens, and tetanus, while the effects of small doses are aggravated by disease of the brain. As a suppository five grains are sometimes prescribed; but I have known this quantity to produce alarming symptoms in a healthy adult. In a case which occurred in London in 1838, a man aged forty-five was killed by ten grains of solid opium; and in September 1843, a woman, aged thirty-eight, was killed by eight grains of the drug given in two doses. (Brit. and For. Med. Rev., Oct. 1844, 558.) In another instance, a lady, aged twenty-nine, suffered from vertigo, numbness of the limbs, and other serious symptoms, when only *twenty drops* of the tincture were introduced in the form of enema, *i. e.*, about one grain of Opium. The same dose had been administered for six nights previously, without any serious effects following;—this fact renders it not improbable that the drug possesses an *accumulative* power. She continued in a state of delirium for twenty hours, and the numbness of the limbs only ceased after forty-eight hours. This was an unusually small dose thus to affect an adult. Dr. Babington has communicated to me the case of a lady who had taken five grains of Dover's powder, *i. e.* about *half a grain of opium*, and who suffered from stupor and drowsiness for three days. These serious effects produced by small doses on adults, must however be considered as exceptions to the rule: they appear to be due to idiosyncrasy, or to a peculiar susceptibility of the poisonous effects of opium in certain constitutions. It must not be forgotten, however, that they lead to one important inference in legal medicine, namely, that an adult may be killed by a dose of opium, which many, relying upon a limited experience, would pronounce to be innocuous. We have commonly no means of detecting or recognising the existence of this idiosyncrasy in individuals prior to the fatal event.

In a case communicated by Dr. Brown to Dr. Christison, four grains and a half of opium, mixed with nine grains of camphor, killed a man in nine hours with all the symptoms of narcotic poisoning. The *smallest dose of solid opium* which has been known to prove fatal to an adult, was in a case reported by Dr. Sharkey of Jersey. (Med. Gaz., xxxvii. 236.) A stout muscular man, æt. thirty-two, short-necked and plethoric, swallowed two pills, containing each about one grain and a quarter of extract of opium,—a quantity equivalent to *four grains* of crude opium: he was soon afterwards attacked by a convulsive fit, and died. He took the opium after having made a full meal. On inspection, there was great lividity of the neck: a large quantity of blood flowed from the scalp, superficial veins, and sinuses of the brain, and there was effused blood, both fluid and coagulated, around the medulla oblongata. There were patches of ecchymosis on the mucous membrane of the stomach: the heart was flaccid, pale, and nearly empty: the vena cava and venous system were much gorged. The plethoric condition of the patient probably tended in this case to aggravate the effects of the opium. The *smallest fatal dose of the tincture* in an adult, which I have found recorded, is *two drachms*. The case is reported by Dr. Skae. (Ed. Med. and Surg. Jour., July 1840.) The patient was a robust man, aged fifty-six;—he swallowed the tincture at ten in the evening, and died under the usual symptoms the following morning; the case thus lasting only twelve hours. The quantity actually swallowed, however, appears to be involved in some doubt; for it is subsequently stated (p. 135) that *half an ounce* of laudanum may have been taken. One fact was ascertained by Dr. Skae, of some medico-legal importance;—that the individual rose from his bed and moved about at least two, and probably three hours, after having taken the poison (see ante, p. 469,) showing thereby that stupor had not supervened at this time. Opium, as meconate of morphia, was detected in the stomach. In another instance, in which the quantity taken was probably equally small, and ultimately proved fatal, the patient was able to converse cheerfully and readily with a neighbour two hours after she had swallowed the poison. In a case which I lately had to investigate, a woman died in twenty-two hours after taking *half an ounce* of tincture of opium by mistake for tincture of rhubarb.

Recovery from large doses.—Very large doses of the tincture have been frequently taken without proving fatal. At St. Thomas's Hospital, a few years since, two women were brought in, who had each swallowed an ounce of the tincture: they both recovered. (Med. Gaz. xix. 264.) Several similar cases have occurred at Guy's Hospital. In July, 1841, a man was admitted, who had swallowed one ounce and a half of the tincture. Vomiting came on, but he was not brought to the hospital until twelve hours after he had taken the poison. The stomach-pump was employed, and even under these disadvantageous circumstances he recovered. Neither the matter then vomited, nor the liquid brought off by the instrument, contained any trace of opium or of meconate of morphia, although the quantity examined amounted to six ounces. About the same time a woman was brought in, who had swallowed ten drachms of laudanum by mistake for tincture of rhubarb. Vomiting ensued, and she did well. It appears reasonable to attribute these recoveries from large doses to speedy vomiting, or to the treatment employed: but this explanation will not always be applicable. A case occurred to Dr. Young (Med. Gaz. xiv. 655,) in which a young lady took an ounce of laudanum in whiskey, and recovered in five days: there was no vomiting, and the cause of the symptoms was not even suspected until she had recovered from her stupor, and confessed that she had taken the poison. Another case occurred at the Westminster Hospital, in December 1843 (Lancet, Dec. 1843,) in which a woman, aged twenty-five, was brought into that institution while labouring under symptoms of poisoning by opium. She was perfectly comatose, the features devoid of expression, the lips purple, and the pupils contracted to the size of a pin's head. The eyes were everted and fixed. Sulphate of zinc and tartar emetic were given without effect, and the stomach-pump was not brought into use until about an hour after her admission. The contents of the stomach were entirely free from the smell of opium. The woman was kept roused, coffee was administered, and she recovered. It was ascertained that she had swallowed one ounce of laudanum, but at what time before her admission, is not stated. It is difficult to say on what the recovery of this woman depended; for a very long time had elapsed before the contents were removed from the stomach, and then there was no trace of opium to be perceived by the smell. Two cases of recovery in young men are reported by Mr. Kirby, in which an ounce and a half of the tincture had been swallowed. They were seen immediately, and owed their recovery to energetic treatment. (Dub. Med. Press, Dec. 24, 1845, 407.) The following instance of recovery from an excessive dose of opium, was privately communicated to me a few years since by one of my class. A medical student, after a hearty supper, at nine o'clock in the evening, swallowed *four ounces* of tincture of opium (P. L.,) made by himself from opium procured at a respectable druggist's. He went to bed and slept until six o'clock the next morning, when he was awakened by a feeling of nausea. He then vomited freely; and, as he supposed, the whole of the contents of the stomach, smelling strongly of opium, were ejected. He perfectly recovered without feeling any other symptom than inability for muscular exertion. This is the largest dose which I have ever known to be taken without fatal consequences ensuing; and it is remarkable, that the opium should have remained so many hours on the stomach without causing serious symptoms and death.

Conditions which modify the effects of opium.—There are some facts connected with the fatal doses of this poison in adults which deserve consideration. The effects of the drug vary with age, idiosyncrasy, habit, state of health or disease, and mode of combination. The aged and the young are much more strongly affected by opium than adults, and very small doses will suffice to kill individuals at the extremes of life. With respect to idiosyncrasy, (see page 37, ante,) its influence over the properties of this drug might be illustrated by numerous cases. Some persons cannot bear the smallest doses of opium without suffering from the most intense headach, and other painful symptoms. Dr. Christison states that he is acquainted with a gentleman who, even when in a state of perfect health, cannot take seven drops of laudanum (*one-third* of a grain of opium) without being narcotized. Two instances have just been mentioned where half a grain and one grain respectively occasioned alarming symptoms in adults. The following is another instance of more recent occurrence, in a case reported by Dr. Steinthal. (Casper's Wochenschrift, Mai 1845, p. 294.) A man who had been suffering from piles, was ordered a starch-clyster containing only *one grain* of pure opium. In about half an hour after its administration the patient was found in a partial state of narcotism. There was pain in the head, which felt hot, the eyes were red and suffused, glassy-looking, and half open; the face flushed; tongue dry; and there was a painful tenderness both in the head and face on the slightest motion. The man was talkative and incoherent; still having sufficient sense to put out his tongue when desired. There was complete loss of power in the upper and lower extremities, and the man was in a half-stupefied condition. A strong cup of coffee (half an ounce to a cup) roused him for a time; but in a quarter of an hour symptoms of narcotism again appeared, and continued for two hours. The man fell asleep, and awoke somewhat refreshed, but he had still lost all power of exerting himself: he slowly recovered from the effects of the dose. It has been suggested that opium exerts a greater proportional effect administered by the rectum than by the stomach, but there is nothing to support this view; on the contrary, the results of practice are decidedly adverse to it. Cases in which this increased effect has been observed may be of an idiosyncratic nature.

From these facts it is obvious that a very small dose, *i. e.* from two to three grains, would in such cases suffice to kill an adult: and death might possibly follow from the effects of one grain. This inference must not, however, be strained too far: before it is admitted there should be some good reason from other sources to believe that idiosyncrasy exists,—a fact which may be commonly ascertained with respect to adults.

Some remarkable results connected with the effect of *habit* on the fatal dose of opium, have been elsewhere detailed. (See page 36, ante.) It is popularly known that opium-eaters will bear very large doses with impunity. It would take a dose of some ounces to kill such individuals. The Turks begin with from one to two or three grains, and increase the quantity gradually until it amounts to two, three, or in many instances six drachms. From three to eight ounces of laudanum are set down as the regular daily allowance of an English opium-eater. The following case is mentioned by Mr. Kirby. A gentleman, of a very haggard, sallow, gloomy appearance, and trembling all over, entered a druggist's shop and asked for an ounce of laudanum, which was properly refused without medical sanction. He then asked to be shown how much an ounce was, and when it stood before him, measured in a graduated glass, he seized it from the counter, and eagerly swallowed it. The druggist was struck powerless. The gentleman, seeing his great alarm, assured him it was needless, as he had been a long time an opium-eater, and took, twice daily, as much as he had just drunk. He remained for half an hour in conversation with the druggist, who was surprised to observe the wonderful changes which so short a period had made in his visiter, whose manner became animated, at the same time that his face became plump and suffused with a pleasing blush. (Dub. Med. Press, Dec. 24, 1845, p. 407.)

Certain forms of disease are observed to aggravate the effects of this drug, and to increase its poisonous properties. Thus it would be dangerous to employ it where there are any symptoms indicative of fulness of the vessels of the brain. In certain chronic diseases of the lungs, attended with cough and expectoration, especially in aged persons, opium in ordinary or even in small doses has been observed to have a very dangerous effect: it tends to produce asphyxia. This fact is of importance, because in the first place it may explain why death occurs from a small dose, and secondly it will exonerate the dispenser of the drug from the charge of having made a mistake, or given an overdose. Medical men ought to be aware of this singular influence of disease: by the want of attention to it they may expose themselves to a charge of malapraxis. There are doubtless other diseases which exert a similar influence, but they are unknown.

On the other hand, disease sometimes exerts a contrary influence. In colic, delirium tremens, hydrophobia, and tetanus, there is a remarkable insensibility to the operation of this drug, owing to the excited state of the nervous system. In the two last-mentioned diseases, opium, in order to produce any effect whatever, is often given in doses which would be certainly poisonous to the individual in health.

Lastly, the operation of opium is much modified by combination with other substances. Its narcotic properties are diminished by combination with tartar emetic, ipecacuanha, camphor, astringent powders, and aromatics.

Action of opium on infants.—As connected with this subject, it is important for a medical jurist to bear in mind, that *infants* and young persons are liable to be killed by very *small doses of opium*; they appear to be peculiarly susceptible of the effects of this poison. Dr. Ramisch, of Prague, met with an instance of a child four months old, that was nearly killed by the administration of one grain of Dover's powder, containing only the tenth part of a grain of opium;—the child suffered from stupor and other alarming symptoms. The following case occurred in June, 1832. Four grains of Dover's powder (containing less than half a grain of opium) were given to a child four years and a half old. It soon became comatose, and died in seven hours. Death was referred to inflammation of the throat, and the jury returned the usual unmeaning verdict of "Died by the visitation of God;" but there was no doubt from the evidence that death was caused by the opiate medicine. Dr. Kelso also met with an instance where a child, nine months old, was killed in nine hours by four drops of laudanum, equal to only *one-fifth part of a grain of opium*: it was much convulsed before death. A case is referred to in a late number of the Medical Gazette, in which two drops of laudanum, equal to the *tenth part of a grain* of opium, killed an infant. The following is a more recent illustration of the fatal effects of a similar dose. A nurse gave to an infant, five days old, *two drops* of laudanum, about three o'clock in the morning. Five hours afterwards the child was found by the medical attendant in a state of complete narcotism. It was revived by a cold bath, but a relapse came on, and it died the same evening, about eighteen hours after the poison had been given to it. On inspection, the brain and abdominal viscera were found in a perfectly healthy state, and there was no smell of opium in the stomach. (Prov. Med. Jour. Oct. 28, 1846, p. 519.) The fatal dose here, as in the former case, was equal to the tenth of a grain of opium, and to only an infinitesimal dose of morphia!

Another case is reported (Lancet, Feb. 1842) in which a child, two days old, was killed by a dose of a mixture containing one minim and a half of tincture of opium, equal to the *twelfth part of a grain*. The child was seized with narcotism, followed by coma, and died in fourteen hours. There are no appearances in the body under these circumstances to indicate the cause of death, and it is not very likely that the poison, when taken in so small a quantity, could be chemically detected by the odour or tests. Dr. Pereira saw a case in which a powerful effect was produced upon an infant by one drop of the tincture of opium, (Op. cit. ii. 1773;) and Dr. Merriman met with an instance where a child, a month old, was thrown into excessive stupor by a single drop of the tincture (equal to the *twentieth part of a grain* of opium) contained in a dose of mixture prescribed for it. He met with two instances where death was caused by a very small dose of Godfrey's cordial, which contains opium. In 1837, an inquest was held in this city on an infant, aged four months, which was killed by half a teaspoonful of Godfrey's cordial. It was properly stated by the medical witnesses, that the proportion of opium in this instance was not more than half a grain to an ounce; and one professed himself ready to swear, that half a teaspoonful could not have caused the death of the child. It is obvious, from what has been said above, that great caution should be exercised in expressing an opinion as to the quantity required to destroy the life of an infant. Six cases are quoted in the Provincial Journal, (April 8, 1846, p. 163,) which show the serious effects of small doses on young children: two of these proved fatal. We cannot measure the effects of opium on infants, by what we observe in adults: but still we find, that in spite of the accumulation of numerous facts, like those above related, there is often a disposition in medical witnesses to refer the death of a child in such cases to natural disease, because the quantity of opium taken, happened to be small. As a proof of this may be mentioned the following case. A woman gave a child four weeks old a narcotic draught, containing *an eighth of a grain* of opium and as much hyoscyamus. The child fell fast asleep, and died comatose in twelve hours. Contrary to well-ascertained facts, the physician declared that the child could not have been poisoned by so small a dose! A case was tried at the Chester Summer Assizes, 1847 (*Reg.* v. *Deays*,) in which this question was put to a medical witness. He was asked whether *four drops* of tincture of opium would kill an infant, and properly replied in the affirmative. The mother was charged with the murder, but acquitted. The learned judge on this occasion held, that the careless or negligent administration of the drug by a mother could not be manslaughter, even although the dose might prove fatal. If she gave it with intention to injure the child, then it would be murder; otherwise she must be acquitted. The same medicine given carelessly by a nurse or stranger, might justify a verdict of manslaughter.

Dr. J. B. Beck has lately published in the New York Journal of Medicine, some excellent remarks upon the effects of opium on the infant subject. He shows that while this drug has a much greater influence on an infant than on an adult, in consequence of the greater impressibility of the nervous system, it is at the same time much more uncertain in its operation; and it is thus liable to prove fatal in very small doses. Among the instances which he has accumulated, illustrative of the powerful action of the drug, he mentions one where a young child was narcotized by fifteen drops of paregoric-elixir, equal to the *sixteenth part of a grain* of opium. This essay has been republished in the Medical Gazette for March 1844, (vol. xxxiii. p. 767.) A case will be presently related, in which, under judicious treatment and adverse circumstances, a child of nine months recovered after having swallowed twenty-five minims of laudanum (p. 486.)

Effects of divided doses.—The quantity of this poison required to destroy life may present itself to a medical jurist as a question under another form; namely, whether a quantity, which, if taken at once would suffice to kill, will prove equally fatal when administered at certain intervals in divided doses. This question is especially important in relation to the effects of opiate mixtures or powders on young children. There are but few facts on record in reference to it; but judging from the known operation of other poisons,—from one case of poisoning by opium, and another by muriate of morphia,—it appears to me certain, when the intervals at which these divided doses are given are so short that the patient has scarcely recovered from the effects of one before another is administered,—that the poison may destroy life when the whole quantity has been taken, although each dose individually might have been harmless. If a sufficient time has elapsed between the doses, for the patient to recover entirely from the effects, there may be some doubt whether death would follow; although it cannot, I think, be denied that opium may possess an accumulative power. (See case, ante, p. 479; also Pharm. Journ., April 1845.) The whole quantity taken at once, might kill in a few hours; while, in divided doses, it might not prove fatal until after a longer period. (See case, post, MURIATE OF MORPHIA.) The nature of the symptoms, as well as the time of their occurrence after taking the medicine, and their aggravation after each dose, are the facts upon which a medical jurist should chiefly rely in forming an opinion as to the cause of death. The post-mortem appearances in the body seldom furnish any information; and the poison is never likely to be discovered in the stomach, since if it exist at all, it can only be in minute fractional parts of a grain. It need hardly be observed, in reference to this question, that when the quantity of poison taken at one dose is not sufficent to kill, it is not likely that death will follow from the medicine being given at distant intervals in divided doses.

PERIOD AT WHICH DEATH TAKES PLACE.

It has been remarked that most cases of poisoning by opium prove fatal in from about six to twelve hours. Those who recover from the stupor, and survive longer than this period, generally do well; but from some cases which have occurred, it would seem that there may be a partial recovery, and afterwards a relapse, (ante, p. 470.) The symptoms, however, generally progress steadily to a fatal termination, or the stupor suddenly disappears, vomiting ensues, and the individual recovers. Several instances are recorded of this poison having destroyed life in from seven to nine hours. One has lately occurred within my knowledge in which an adult died in five hours after taking the drug prescribed for him by a quack. Dr. Christison met with a case which could not have lasted above five, and another is mentioned by him which lasted only three hours. Dr. Beck quotes a case which proved fatal in two hours and a half. (Beck, Med. Jur., 873.) The most rapid case of death yet reported was that of a soldier who was accidentally poisoned, in September 1846, in the Hospital of Val de Grace. It appears that he swallowed by mistake about an ounce of laudanum, and died in convulsions in *three-quarters of an hour* afterwards. (Journal de Médecine, Octobre 1846, p. 475.) It is possible that the drug may kill even with greater rapidity than this; but as a medico-legal fact, we are at present entitled to state, that it has destroyed life within the short period above mentioned. On the other hand, the cases are sometimes much protracted. There are several instances of death in fifteen or seventeen hours. I have known one case fatal in twenty-two hours, and among those collected by Dr. Christison the longest lasted twenty-four hours. (Op. cit. 712.) In eight fatal cases of poisoning by opium reported by Dr. Beck, the smallest quantity taken was one drachm,—the largest, one ounce and a half. The shortest time between the taking of the poison and death was eight hours,—the longest twenty hours,—the average time of six cases fourteen hours. (Dub. Med. Press, May 1845.) In the same journal it is stated, on Dr. Beck's authority, that out of thirty-nine fatal cases of poisoning by laudanum, the smallest quantity taken was one drop, the largest sixteen ounces. The shortest period for death was *two hours*, the longest *forty-eight hours;* average of twenty-three cases, twelve hours. These cases lose their interest, as the details are not quoted.

TREATMENT.

The first object is to remove the poison by the stomach-pump, or in the case of an infant, by a catheter, as speedily as possible. This instrument should be employed until the water used for washing out the organ has no longer the colour or smell of opium. The entire absence of the drug may be perhaps better indicated by adding to the liquid a few drops of a solution of a persalt of iron. If no red colour be produced, there is reason to believe that there is no meconic acid, and therefore no opium, present. (See tests for MECONIC ACID.) In thus removing the poison, we at once arrest the progress of absorption. Emetics are of no service unless the individual possesses the power of swallowing. Occasional doses of sulphate of zinc may then be given to him, and in the intervals, a decoction of strong coffee or tea. Cold affusion on the head, chest, and spine, has been adopted with great success, and in infants the plunging of the body into a warm bath, and suddenly removing it from the water into the cold air, has been found a most effectual means of rousing the child. (Med. Gaz. xxv. 878.) [Many cases are also recorded by Dr. Cross, Transylvania Journal, i. 469.—G.] Flagellation to the palms of the hands and soles of the feet or the back, has also been successfully employed. A common way of rousing an adult is to cause him to keep in continual motion, by making him walk between two assistants. Above all things, the tendency to fall into a state of lethargy must be prevented. If called to a person already in a lethargic condition, the application of shocks to the head and spine by an electro-magnetic apparatus will be found most effectual. It has in several instances led to the recovery of an individual when in an almost hopeless condition. (See p. 74, ante.) As an illustration of the effects of an overdose of this drug on young children, and of the benefits derivable from the electro-magnetic treatment, the following case, reported by Mr. Colahan, will be found of some interest. (Dub. Med. Press, April 22, 1846, 244.)

A healthy child, aged nine months, had given to it by its mother, at one o'clock A. M., twenty-five minims of laudanum to procure sleep. The laudanum was examined by Dr. Christison, and found to be of the usual strength. A short time after its administration the mother's attention was attracted to the child from its unusually loud breathing. The child was then applied to the breast, which it received with its usual avidity, after which it was placed in bed, when natural sleep was supposed to be enjoyed, and so continued undisturbed for six hours. At seven A. M., the patient's respiration becoming more oppressed, and, as it was not sensible to impressions after being removed from bed, the mother called on Mr. Colahan to see her child, which she said had been ill since the previous night. At eight A. M., it presented the following appearances:—Countenance pale and placid, with an expression of deep and perfect repose; eyelids closed: it appeared to be in a peculiar slumber, from which, when aroused, it evinced a constant tendency to sleep; the respirations were hurried, at times stertorous, and occasionally accompanied with distinct *stridor*, with signs of intense bronchial irritation, and copious mucous secretion without expectoration; pulse rapid and distinct, with hot skin. The child had been under the influence of laudanum for seven hours and a half, when emetics, flagellation, and cold affusion, were resorted to. Vomiting was excited with evident relief, and renewed by the cold affusion. This state of restored consciousness was always imperfect, and was speedily followed by lethargy and profound coma when the stimulating influence was withdrawn. The pupils were contracted, and the retina was insensible to the stimulus of light. This treatment was continued for two hours incessantly, when sensibility, although imperfect, was easily excited. At one P. M., the insensibility and torpor had evidently increased, with signs of cerebral congestion. The previous excitants,—the internal use of ammonia, &c. &c., with inhalation of its vapour, now seemed useless, and signs of revival were with difficulty produced. Congestion with consequent sopor increasing, with an evident decrease of vital energy, the child was brought to the hospital, and subjected to the action of an electro-magnetic coil apparatus. The strength of the electric current was at first comparatively feeble; the shock caused by its influence, although perceptible, was slight, and proportionally diminished after application to the different sensitive parts of the body. The strength of the battery was now increased by sulphuric acid, and a rapid current of electricity developed, to the powerful stimulus of which the infant was exposed, alternately applied to different parts. The physiological effect was now apparent; and it was not until the regulator was moved to its highest point, and the flow of electricity had reached its maximum intensity, that complete signs of revival were produced, causing involuntary muscular contractions; with frequent voluntary efforts of the child to get released from its painful position. The galvanic influence was continued for five hours, by which time sensibility returned, with restoration of the functions of the nervous centres. Gradual and progressive recovery continued, and in a few days the infant was perfectly convalescent.

[Dr. Lee also speaks highly of this agent, in his notes to Guy's Forensic Med. 668.—G.]

In a case in which two ounces and a half of laudanum had been taken, and the patient refused to submit to the usual treatment, an emetic was injected per anum, and it is reported, with success. (Med. Gaz. viii. 12.) Artificial inflation of the lungs has been employed in the lethargic state, but it is of doubtful benefit, since the great object is to apply a direct stimulus to the nervous system. Bleeding should not be resorted to until *after* the poison has been completely removed from the stomach, as the abstraction of blood acts injuriously by tending to promote absorption. It is, indeed, only justifiable when, during the second stage, there is a strong and full pulse, with symptoms indicative of cerebral congestion. If the pulse should become feeble, and sink after the abstraction of a small quantity of blood, the operation should be carried no farther. There are very few cases in which bleeding is necessary, and, as Mr. Bullock has remarked, its tendency is rather to protract recovery. (Med. Gaz. xix. 264.) Its indiscriminate application is decidedly injurious. When there are signs of recovery, ammonia applied on rags to the nostrils, and frictions to the chest with the compound camphor liniment, will aid in restoring the patient. Cataplasms of mustard and cayenne pepper have been applied with advantage. The means above stated, variously employed, have been found eminently successful; but especially the removal of the poison by the stomach-pump. Out of numerous cases of poisoning by opium, brought to Guy's Hospital, but very few have proved fatal, even when the remedial treatment was applied late. Antidotes are of little avail. (See p. 74, ante.) Vinegar was formerly recommended, but it is known to be a powerful solvent of morphia and other poisonous alkaloids. Its use, therefore, has long been abandoned as decidedly objectionable. A case of poisoning by laudanum has been lately reported, in the treatment of which vinegar was given in two-ounce doses every half-hour. The effect was to increase the stupor, and other bad symptoms. (Med. Gaz. xxxviii. 683.) Orfila has recommended *tannin* in the form of galls, oak bark, cinchona, or very strong black tea, as a chemical antidote, because it has the property of precipitating morphia from its solution in the form of a comparatively insoluble tannate. If this substance has acted beneficially, it has most probably been by its stimulant and astringent properties. At any rate, as the tannate of morphia is not more insoluble than the pure alkaloid itself (an active poison,) which requires 1000 parts of water to dissolve it, and it is perfectly soluble in the acid secretions of the stomach, it is difficult to admit Orfila's chemical explanation.

The best liquid for exhibition, is undoubtedly a very strong decoction of coffee. This operates as a safe and excellent stimulant when the power of deglutition is retained.

It has been long known that the vegetable alkaloids underwent decomposition by chlorine, and M. Flandin has recently announced that when morphia dissolved in a weak acid is combined with any alkaline hypochlorite, it is liable to be decomposed and rendered inert. He suggests, therefore, that acetic or any other weak acid might be safely used in poisoning by opium or morphia, provided it was combined with one of those salts. (Gaz. Méd. Juillet 31, 1847, 620.) It appears to me that it would be decidedly objectionable to administer as an antidote a hypochlorite with any acid (p. 233, ante.) We might as well exhibit chlorine at once; and as to the beneficial operation of such a mixture, this remains yet to be established by experiment.

In a late number of the Gazette Médicale, M. Bouchardat has recommended the employment of the ioduretted iodide of potassium as an antidote. According to him, this solution forms insoluble and inert compounds with the alkaloids, especially with morphia. The product, he states, is even insoluble in weak acids. The proportions which he recommends are, iodine three grains, iodide of potassium six grains, water one pint. It is recommended to be given in doses of a wine-glassful, and vomiting should at the same time be promoted by emetics. (Janvier 9, 1847, p. 31.)

POISONING BY POPPIES.

The heads of the white poppy, grown in this country, contain meconate of morphia. They yield an inspissated extract called English opium, which, according to Mr. Hennell, contains five per cent. of morphia. The white poppy-heads, therefore, yield to water in the form of decoction, a poisonous salt capable of acting deleteriously on young children. Many cases of poisoning have occurred from the injudicious use of *Syrup of poppies*, which is nothing more than a sweetened decoction of the poppy-heads. This syrup is said to contain *one grain* of extract (opium) *to one ounce* (Thomson.) The common dose of it for an infant three or four months old, is half a drachm,—for adults, two to four drachms. (Pereira, ii. 1769.) There is some reason to believe that what is often sold by many druggists for syrup of poppies as a soothing or cordial medicine for children, is nothing more than a mixture of tincture or infusion of opium with simple syrup; it is therefore a preparation of very variable strength. This may account for what appears to many persons inexplicable, namely that an infant may be destroyed by a very small dose. In January 1841, a child six months old is said to have died from the effects of less than half a teaspoonful of syrup of poppies bought at a retail druggist's. The narcotic symptoms were fully developed in three-quarters of an hour. The syrup in this case may have contained tincture of opium. Seven children are reported to have lost their lives by this syrup in 1837-8. In one of these cases, a teaspoonful and a half was given. Stupor came on in half an hour, and the child died the following day. A teaspoonful has been known to prove fatal to a healthy child. (Pereira, ii. 1769.)

The following are cases of poisoning by the *Decoction of poppies*. A woman boiled two poppy-heads in a quarter of a pint of milk, and gave only two small spoonfuls of this decoction to her child. In an hour, the child fell into a deep lethargic sleep,—the respiration became stertorous, and in ten hours it died. On inspection, the brain and its membranes were found congested. In a second case, a maid-servant in order to quiet a child, gave it two teaspoonfuls of a decoction made by boiling one poppy-head in a small pot of water. The child was found dead in the morning. The brain and its membranes were much congested; and the ventricles contained bloody serum. The seeds of the poppy were found in the stomach.

The effects are not always of this serious character, as the following case will show. A woman gave to her child several teaspoonfuls of a strong decoction of poppy-heads. In a quarter of an hour it fell into a deep sleep, from which it could not be roused. Medical assistance was not called for thirty-six hours, and then the child was almost moribund. The eyes were sunk, the lids half open and surrounded by a livid circle, the pupils *dilated* and insensible, the face pale, with a slight blueish tint, the extremities almost paralyzed, respiration hurried, the pulse frequent, small and trembling, the forehead covered with a cold sweat, and the lower jaw depending. No urine had been voided, nor had the child passed any motion, since the first occurrence of the symptoms. It was too late to think of removing the poison from the stomach. Coffee and other stimulants were used, under which the child recovered. (Ann. d'Hyg. 1845, i. 212; also Med. Gaz. xxxvi. p. 305.)

It may be observed that the poisonous salt of morphia is generally considered to exist in the *capsule* of the poppy, and not in the seeds; but Sobernheim mentions one or two cases of poisoning by the seeds of the plant. (Tox. 500.) For other cases, see Henke Zeitschrift der S. A. 1844, i. 302. *Extract of poppies* acts like the decoction, but it is more powerful. The dose of this medicinal preparation for adults, is from two to twenty grains.

GODFREY'S CORDIAL.

This is chiefly a mixture of infusion of sassafras, treacle, and tincture of opium. The quantity of tincture of opium, according to Dr. Paris, is about one drachm to six ounces of the mixture, or *half a grain of opium to one ounce*, but it is very probable that, like the so-called syrup of poppies, its strength is subject to great variation. A case has been already related (ante, p. 483,) in which half a tea-spoonful, = 1-32d part of a grain of opium, was alleged to have caused the death of an infant. In 1837-8, twelve children were admitted to have been killed by this mixture alone. The explanation of this is, that the medicine is given in large doses by very ignorant persons.

[The formula for Godfrey's cordial adopted by the Philadelphia Coll. Pharm. orders rather more than one grain of opium to the fluid ounce.—G.]

DALBY'S CARMINATIVE.

This is a compound of several essential oils and aromatic tinctures in peppermint water, with carbonate of magnesia and tincture of opium. According to Dr. Paris, there are *five drops* of the tincture, or one quarter of a grain of opium, to rather more than *two ounces* of this mixture, or *the one-eighth of a grain to an ounce*. The formula commonly given is, carbonate of magnesia two scruples, oil of peppermint one minim, of nutmegs two minims, of aniseed three minims, tincture of opium five minims, spirit of pennyroyal and tincture of assafœtida of each fifteen minims, tincture of castor and compound tincture of cardamoms of each thirty minims, and of peppermint water two ounces. According to this formula, tincture of opium forms the 1-211th part by measure, or one teaspoonful would contain the 1-64th part of a grain of opium. Like most of these quack-preparations, it probably varies in strength. An infant is reported to have been destroyed by *forty drops* of this nostrum,—a quantity, according to the strength assigned, equivalent to more than *two drops* of the tincture or one-tenth of a grain of opium. Accidents frequently occur from its use, partly owing to ignorance, and partly to gross carelessness on the part of mothers and nurses.

PAREGORIC ELIXIR. COMPOUND TINCTURE OF CAMPHOR.

This is a medicinal preparation of alcohol, opium, benzoic acid, oil of aniseed and camphor. Opium is the active ingredient, and of this the tincture contains rather less than *one grain* in every *half ounce* (nine grains to five ounces.) It is sold to the public at the rate of fourpence per ounce. Fatal cases of poisoning by paregoric are not very frequent: the following occurred in March 1843. A child between five and six years old, had had some cough medicine prescribed for it, at a druggist's. The medicine consisted as nearly as could be ascertained from a portion left in the bottle,—of paregoric, having about from one-fourth to one-half the strength of the pharmacopœial tincture. The child took two-thirds of this mixture, given in divided doses, at somewhat irregular intervals, and died in about thirty-six hours. The quantity of opium in the portion of the mixture taken, was, judging by comparison with the usual strength of the tincture, from three-fourths of a grain to one grain and a quarter. The child became drowsy after each dose, and slept on one occasion for several hours together. It was occasionally roused, and appeared sensible; but again relapsed into drowsiness on taking the medicine. A few hours before its death, it was found comatose, with stertorous breathing and strongly contracted pupils. On inspection, the whole of the organs were healthy, with the exception of the parietes of the ventricles of the heart, which were somewhat thickened;—there was no turgescence of the vessels of the brain, nor effusion in the ventricles. The liquid contents of the stomach yielded no trace of opium or an opiate. There was, however, no doubt that the death of this child had been caused by an opiate. This was proved, 1, by the nature of the medicine taken; 2, the nature of the symptoms, which were aggravated after each dose; 3, the confirmed coma and stertor; and, lastly, the absence of every other cause to account for the rapid death under the circumstances. (G. H. Reports, April 1844.) It was a question here, how far a small quantity of opium in divided doses was likely to prove fatal to a child of this age? The answer was given to the effect, that although each dose might be individually harmless, the frequent repetition of the medicine, when the child had scarcely recovered from the effects of the former dose, might operate fatally. (See p. 484, ante.)

In another case, a child aged 7 months was killed by a tea-spoonful given in two doses at an interval of a day, *i. e.* by a dose equal to one quarter of a grain of opium. (Pharmaceut. Jour., Ap. 1845, p. 464.)

DOVER'S POWDER. (PULV. IPECAC. COMP.)

This is a preparation of opium, the effects of which on young children have been already adverted to (ante, p. 482.) The proportion of opium is one-tenth part, or *one grain* in every *ten grains* of the powder. A child has been killed by four grains—therefore by a quantity containing about two-fifths of a grain of opium.

The following case of poisoning by Dover's powder is reported by Mr. Griffiths. (Medical Gazette, March 1844.) About ten grains of the powder were given by mistake to an infant seven weeks old, and it died in twenty-four hours. On an inspection of the body, the countenance was placid, and the fingers of both hands were firmly contracted. In the abdomen, the colon was seen to be distended with flatus; the spleen, kidneys, and intestines were healthy; the liver gorged with blood; the stomach contained a very small quantity of colourless viscid matter. The inner coat was vascular; and at the great curvature as well as in other parts, the blood-vessels were highly injected in patches. The lungs were gorged with blood; the upper lobes being infiltrated with a greenish serum. The pericardium was vascular, and contained about a drachm of fluid. The right auricle was empty; the left ventricle contained some thin fluid blood, and a small coagulum. The sinuses of the dura mater were filled with dark coagula; the surface of the brain appeared covered by a complete network of vessels, distended with light-coloured blood. On the surface of each posterior lobe of the cerebrum, slight extravasation had taken place. The brain was soft, and the difference of colour between the gray and white matter barely discernible. The vessels in the substance of the brain were gorged with blood, presenting, on section, a thickly studded appearance—the spots were of a deep dull red, and in many places coalescing. There was a small quantity of fluid in each lateral ventricle, and on the floor of each, the blood-vessels were largely distended. There was serous effusion on the surface as well as at the base of the brain, to the amount of about half an ounce. The contents of the stomach were carefully analyzed, but neither morphia nor meconic acid could be detected. This case is interesting in several particulars. In the first place, it is surprising that so young an infant should have lived so long after taking a dose equivalent to *one grain* of opium. Making every allowance for the great vascularity of the brain in young subjects, it appears from the inspection, that the opium had here affected the organ, and caused a general congestion as well as effusion and slight extravasation;—the last-mentioned condition is somewhat rare in poisoning by opium. The non-detection of the poison in the contents of the stomach was sufficiently accounted for by the very small quantity of opium in the dose of Dover's powder, and by the length of time which the child survived. The opium contained in ten grains of Dover's powder is equivalent only to about the twentieth of a grain of morphia, and probably about the same proportion of meconic acid. It has been already observed that it is extremely rare to find opium in the stomachs of young children poisoned by small doses.

OPIUM LOZENGES.

It is not generally known that cough lozenges sold to the public often contain a large quantity of opium. Mr. Garlick has communicated to the Lancet a case in which he was called to a man who had occupied himself during an afternoon in sucking one ounce and a half of these lozenges. After a time he was observed to become drowsy. His countenance was very pale: there was great somnolency; and it was with difficulty that any reply to a question could be obtained. The pupils were strongly contracted; the breathing heavy and oppressed, occasionally stertorous; and the pulse small and feeble. With some difficulty, and after active treatment, the man recovered. For twenty-four hours afterwards he experienced general numbness. The vender of the lozenges knew nothing about the quantity of opium contained in them! (Lancet, 137, Jan. 30, 1847.)

[Wistar's Cough Lozenges, so much employed in this city, contain about 1·60 of Opium.—G.]

MORPHIA AND ITS SALTS.

Morphia and its saline combinations must be regarded as active poisons. The pure alkaloid is known from its salts by its great insolubility in water, (see post, p. 501) and owing to this property some have regarded it as less poisonous. The acrid secretions of the stomach would, however, dissolve it in sufficient quantity to produce very speedily dangerous effects. The two principal salts of morphia are the MURIATE and the ACETATE. The first of these is now largely employed in medicine, and both are preferred to the pure alkaloid, which is seldom given medicinally. They are used in powder and solution, but most commonly in the latter form. The medicinal dose of morphia, or of either of its salts, may be stated to be from one-eighth of a grain gradually increased to two grains. There is no other poisonous substance so completely under the influence of habit,—hence very large doses may be borne after a short time with impunity. When applied endermically, the cuticle having been previously removed by a blister, a dose of from one to two grains is sprinkled over the skin: but violent symptoms have been occasionally observed to follow even from *half a grain*. The Muriate of morphia is used, according to the Edinburgh Pharmacopœia, dissolved in rectified spirit and water,—the solution being of such a fixed strength as to be equivalent to tincture of opium. One ounce of the solution contains *four grains and a half* of the muriate, and the dose for an adult is from twenty to forty minims. It is important that the medical practitioner should be well acquainted with the strength of morphia and its preparations compared with the ordinary preparations of opium (see ante, p. 466.) Morphia and its salts have an intensely bitter taste.

SYMPTOMS.—The case of *Dr. Castaign*, in 1823, (see post, p. 494) first called the serious attention of the profession to the poisonous properties of the salts of morphia. Soon after the trial and conviction of this person, numerous monographs on this subject appeared, and many experiments were performed in order to determine not only the ordinary effects of the salts of morphia, but the best process for its detection in the dead body. Dr. Vassal describes the symptoms produced by Acetate of morphia used medicinally in the following order:—1, an irresistible tendency to sleep; 2, a well-marked slackening of the circulation, the pulse becoming small and slow, but remaining regular; 3, the contractility of the pupils, as well as the sensibility of the retina augmented; 4, copious perspiration during sleep—this ceases if the use of the salt be continued, but reappears on every increase of dose; 5, obstinate constipation; 6, more or less retention of urine; 7, irritation over the whole of the skin, accompanied in some instances by an anomalous eruption. (Considérations Médico-Chimiques sur l'Acétate de Morphine, 102.) When either of the preparations is taken at once in an overdose, the symptoms are more strongly marked, and they follow each other more speedily. They generally *commence* in from *five to twenty minutes* after the dose of poison has been swallowed; and they very closely resemble those observed in poisoning by opium. As a summary, it may be stated, that they consist in dimness of sight, weakness and relaxation of the muscular system, tendency to sleep, stupor, loss of consciousness, coma, stertorous respiration, and more commonly than in poisoning by opium, there are convulsions. According to Orfila, in nineteen-twentieths of all cases, the pupils will be found strongly contracted, a statement which I believe to be correct: the few exceptional cases were those in which the dose was excessive, and the symptoms were unusually violent. The state of the pupils gave rise to great difference of opinion among the medical witnesses on the trial of Dr. Castaign. (Orfila, ii. 185; see post, p. 495.) The condition of the pulse varies greatly. In some cases there is great irritation with itching of the skin, and irritability of the bladder with difficulty of passing urine. Vomiting and diarrhœa have been met with in those instances in which the dose was large.

POST-MORTEM APPEARANCES.—The only post-mortem appearance which can be referred to the action of morphia, or its salts, is fulness of the cerebral vessels, with occasionally serous effusion. These poisons have no local irritant action, and they, therefore, leave no marks of their operation in the stomach and bowels.

QUANTITY REQUIRED TO DESTROY LIFE.—One of the most important questions connected with this form of poisoning, is that which relates to the dose of morphia required to destroy the life of an adult. Although from facts to be presently stated, there can be no doubt that the salts of morphia are very energetic poisons, several cases might be quoted to show that they are sometimes taken with impunity in large doses. The conditions of the body which influence the effects of opium (ante, p. 481,) operate equally with respect to morphia. The MURIATE of MORPHIA has been given in doses of *two grains* every six hours without ill effects; but then the commencing dose was a quarter of a grain, and this was gradually increased. Under these circumstances, it is well known that after a time, as much as from eight to ten grains will be required to act as a narcotic. In diseased conditions of the nervous system, what might be regarded as poisonous doses of these salts are given without injury. Dr. Pereira states, that in a case of insanity he gave *two grains* of the muriate at one dose, without any ill effect following. (Op. cit. ii. 1779.)

In one instance that came to my knowledge, an adult, who had taken the muriate medicinally, was killed by a dose of three grains. In a case which occurred in London, in April 1846, a woman, aged sixty-six, was killed by a dose of six grains, under the following circumstances:—She was admitted into University College Hospital, labouring under gangrene of the right foot. A draught containing three quarters of a grain of muriate of morphia was prescribed for her: she had taken this dose, it appears, safely on two or three previous occasions. By some mistake, a stronger solution of morphia than that intended by the prescriber was used, and a draught, containing *six grains* of the muriate, was given to her by the nurse about nine o'clock. In three hours, the woman was found in a state of complete narcotism, and in spite of every available remedy, including galvanism, she gradually sank, and died *nine hours* after taking the draught. (Med. Times, May 9, 1846, p. 114.) Dr. Christison has collected three cases in each of which ten grains of the muriate proved fatal. In one of these, a woman took this dose of the pure muriate by mistake. This being discovered immediately, means were used to prevent any ill effects from the accident. Within half an hour after the poison had been swallowed, the stomach was completely cleared by the stomach-pump. At this time the patient was quite sensible. Stupor quickly came on,—deep coma gradually appeared; and before night she expired. (Op. cit. 727.) This case is important, because it shows,—first, that *absorption*, to an extent to prove fatal, may occur in *half an hour;* secondly, that there may be a complete remission in the symptoms, and afterwards a fatal relapse, (see ante, p. 469;) and thirdly, it throws an important light upon the case of *Castaign* (p. 494,) as it proves that a person may die after many hours from the effects of one dose of morphia, and no trace of poison remain in the stomach.

The Acetate of Morphia is subject to similar anomalies with respect to dose. Mr. Headland states that he attended an opium-eater, who was in the habit of taking seventeen or eighteen grains of the acetate daily. A case is reported in most works on toxicology, in which a young man swallowed ten grains of the acetate, and shortly afterwards forty grains: he suffered from the usual symptoms, but ultimately recovered, although he had taken in the whole, fifty grains of the acetate of morphia on an empty stomach! An account of this case will be found in Schneider's Ann. der S. A., 1836, i. 455. In the Edinburgh Medical and Surgical Journal, (vol. xxxiii. p. 220,) a case is reported where a young man recovered in four days, after taking *twenty grains* of the acetate; but the most remarkable instance of all, is that reported by M. Bonjean, in which a young man, aged twenty-four, entirely recovered in about eight hours from a dose of *fifty-five grains* of acetate of morphia in an ounce of water. No symptoms of any importance manifested themselves until *an hour* after the poison was taken, and there was then simply vertigo with slight somnolency. Two hours after the occurrence, he still had the power to answer questions! In four hours deep coma supervened, but under copious bleeding and other treatment, this gradually disappeared. (Ann. d'Hyg. 1845, i. 150.) The following is a more recent case of recovery from a large dose:—A man swallowed at once *ten grains* and three quarters of acetate of morphia. Tartar emetic was immediately given, but without producing vomiting. About three hours after the accident, and while the patient was in a state of deep coma, highly concentrated solution of coffee with the solid residue was given to him. The patient swallowed about ten ounces in twelve hours. The coma disappeared, and he perfectly recovered. (Gaz. Méd., Mai 1, p. 346.) On the other hand, from an interesting case which occurred in 1838, (Lancet, Nov.,) there is reason to suppose that only *half a grain* of the acetate of morphia caused the death of a lady to whom it had been administered as a medicine! She was at the time in a state of ill health.

According to Dr. Melion, this salt (acetate of morphia) exerts proportionably a much more powerfully narcotic effect than opium. Even from small doses administered medicinally, he observed the following symptoms:—the child became dull and drowsy, and gradually fell into a state of stupor; it lay with its eyes shut, or half-open—one more so than the other; the ball of the eye fixed or rolling about: the pupil contracted and inactive; the heat of the head increased, and the scalp and face covered with a copious perspiration. The child murmured or spoke during its sleep, and moved its upper lip and jaw as if in the act of sucking; if it awoke, it expressed a desire to drink, and again fell asleep. It continued in this state for eight or twelve hours. In a more advanced stage, there was venous congestion over the whole body, the child lay listless, the skin was purple, the temperature diminished, the pupils strongly contrasted and quite inactive, the cardiac pulsations weak, the respiration slow, the pulse quick or slow, small and weak, and all the secretions and excretions were suppressed. Unless quickly roused, convulsions ensued, and these were followed by death. (Monthly Journal of Medical Science, Dec. 1846, 455.)

On the whole, notwithstanding the anomalies which have been observed, we are, I think, justified in regarding it as rather the exception to the rule, that a person not used to the drug, should escape, who has taken more than *two grains* of either salt, and that a far less quantity than this will suffice to kill a child. Dr. Vassal states, that when he gave in one day more than *three grains* of the acetate to a patient, narcotism was invariably induced. Symptoms of poisoning have certainly been produced by *one grain* of the acetate; and that the muriate given in this dose may destroy life, is proved by the following case reported by Dr. Paterson. A girl, æt. 19, was brought to the Edinburgh Infirmary, in December 1845, labouring under the following symptoms:—She was perfectly insensible; the breathing somewhat hurried and oppressed; pulse almost imperceptible, and so rapid that it could not be counted; the pupils were contracted to the size of a pin's head. It was ascertained that an eight-ounce cough-mixture had been prescribed for her, containing two drachms of the solution of muriate of morphia, equivalent to rather more than *one grain* of the salt. She commenced taking the mixture at 8 P. M., and, being anxious to obtain relief for her cough, she had taken the whole of it in divided doses by 2 A. M., (over a period of *six* instead of *twenty-four hours.*) She then fell into a state of insensibility, which had continued up to the time of her admission to the hospital at 9 o'clock A. M. She died soon afterwards: *i. e.* rather more than thirteen hours from the period at which she commenced taking the mixture. On inspection the only morbid change observed in the head, was a somewhat less-marked appearance of the depressions between the convolutions; and the vessels contained dark-coloured blood. There were marks of inflammatory disease in the lungs. The stomach, which was much corrugated, contained a pint of greyish-coloured fluid. It was perfectly healthy. In the heart and great vessels there were numerous coagula of blood, showing that fluidity of the blood is not an indication of narcotic poisoning. There was purple lividity of the face, neck, and arms. (Monthly Jour. Med. Science, Sept. 1846, p. 191.) The case is interesting, not merely on account of its furnishing an illustration of the *smallest fatal* dose of this poison yet known, but because the dose was taken at intervals during a period of six hours. It corroborates the inference of poisoning by paregoric elixir in a case elsewhere described. (See p. 489.) The frequent repetition of a poisonous medicine, if the interval between the dose be not sufficiently long to allow the individual to recover from its effects, will operate as fatally as if the poison was given at once. It also tends to strengthen the view that the salts of morphia have an accumulative property. Dr. Paterson suggests what is not improbable, that the inflamed condition of the lungs may have added to the intensity of the effects produced by the morphia. We learn from this case that the muriate of morphia is a very energetic poison. Dr. Kelso states that he suffered most formidable symptoms from taking only about *half a grain* of the muriate of morphia. (Lancet, Sept. 1839.)

The poisonous action of the salts of morphia gave rise to an important medico-legal investigation in Paris in 1823, which ended in the trial and execution of a *Dr. Castaign*, who had formerly been a pupil under Orfila, and was charged with having made use of his knowledge of poisons to take away life. The facts were these:—Dr. Castaign was intimate with two brothers, who were young men of fortune. One of them, Hippolyte Ballet, who had been for some time an invalid, and was attended by Castaign, died in October 1822. Although he died rather suddenly and under severe symptoms, no suspicion arose that his death had occurred from other than natural causes. On inspection, nothing was observed in the body to indicate violent death. He made his will in favour of Castaign, and left nothing to his brother. Castaign afterwards surrendered the will to the surviving brother for a large sum of money. About seven months afterwards the prisoner Castaign, who had been travelling about with the surviving brother, Augustus Ballet, unaccompanied by servants, put up at a tavern at St. Cloud. In the evening Ballet complained of feeling unwell: some sugared wine was given to him by Castaign, but without relief. The prisoner left his companion at four o'clock in the morning, as he said to take a walk in the park; but it was proved that instead of doing this, he privately returned to Paris, procured at the shop of one druggist twelve grains of tartar emetic, and at another shop *twenty-six grains* of acetate of morphia. He immediately returned to Ballet, whom he found still lying ill. He prescribed for his patient cold milk, and gave it to him himself. In five minutes Ballet was seized with convulsions, and in about half an hour with vomiting and purging. He was attended by a physician, who treated the case as one of cholera morbus. In the evening the patient became insensible; unable to swallow; bathed in a cold perspiration; with a small pulse; burning skin, jaws locked, the neck rigid, the abdomen tense, and convulsions of the extremities. After a few hours he was seen by another physician: the breathing was then stertorous, and the pupils much contracted: there was an entire loss of sensibility in the legs. He died about thirty-two hours after the commencement of the symptoms in a severe form. On inspection the chief morbid appearance was congestion of blood in the brain, with serous effusion beneath the membranes. The contents of the stomach were analysed by Vauquelin and Barruel, but no trace of any poison could be detected.

Owing to the strong moral evidence against him, the prisoner was put on his trial for the murder of the deceased: and the most eminent medical jurists in France gave evidence on the occasion. The opinions of Orfila, Magendie, Vauquelin, and Laennec, went to show that the symptoms and death might be referred either to vegetable poison or to natural disease. The absence of poison in the body might have been due to vomiting, absorption, and the difficulty of detecting the salt of morphia. Chaussier appeared not so much in the capacity of a medical witness, as a *medical defender* of the accused. When asked whether it was possible that the effects could have been due to vegetable poison, he gave an evasive answer; whether the acetate of morphia might not be absorbed so as to leave no trace—he said that it might, but it required time. Can death follow (*i. e.* from poison) when there has been vomiting?—No, the poison is expelled! Further, he stated that narcotic poisons, including acetate of morphia, always caused a prodigious dilatation of the pupil! (See Smith's Anal. of Med. Ev. 368, and Beck's Med. Jur. 877.)

The other parts of the evidence it is unnecessary to enter into; the trial lasted eight days, and the prisoner was condemned and executed. If this case be regarded only in a *medical* point of view, there was an entire absence of proof that the deceased had died from the effects of acetate of morphia. As in the case of *Donellan*, (see PRUSSIC ACID) the jury appear to have been satisfied of the guilt of the prisoner from *moral* rather than from medical circumstances:—it was proved to them medically, that the symptoms and appearances were such as might be expected to follow a poisonous dose of a salt of morphia; that there was nothing in them inconsistent with this hypothesis, but on the contrary so far as they went they actually supported it;—and that the want of direct medical proofs might easily be explained. There can hardly be a doubt that by the greatest refinement of villany, the prisoner procured and administered the emetic for the very purpose of defeating that investigation which would have clearly established his guilt. The prisoner admitted that he purchased fifteen grains of acetate of morphia, and he wished the jury to believe that his object in procuring it at four o'clock in the morning, under the very suspicious circumstances mentioned, was for the sake of performing some experiments on dogs and cats! Unless, then, a jury be permitted a reasonable latitude in forming an opinion on the *whole* of a case, the most terrible form of perpetrating murder by poison, would be openly tolerated, and the criminal law might be safely set at defiance. It is sad to reflect that the distinguished Chaussier should have shown himself so completely a partisan on this occasion, as to have endeavoured by his evidence to obscure the truth. The replies quoted are perfectly indefensible; because they either do not state the *whole* truth, (the great point of duty in a conscientious witness) or they entirely pervert it!

I am not aware that the salts of morphia have been criminally used as poisons in this country, but it is necessary to state that they are sometimes recklessly prescribed in dangerous doses. The following case, referred to me in July 1847, is instructive in more than one point of view:—A lady of this metropolis consulted a homœpathic physician who had acquired great repute as an advocate of the system of *infinitesimal* doses. He prepared and gave to her some small white powders, with explicit instructions in writing as to the mode of taking them, and the nights on which they were to be taken. She took, as I was informed, two, and on each occasion she suffered from great stupor and all the symptoms of narcotic poisoning, followed by diarrhœa. A suspicion arose that the powders contained some very active ingredient, and three of them were sent to me for analysis. It was first ascertained that although in appearance and from the directions they were intended to pass as equally divided doses of the same medicine, they differed greatly in weight. The first weighed 3·4 grains; it consisted of calomel and morphia, the latter being in the proportion of *one grain!* The second weighed 1·5 grains: it consisted entirely of *sugar of milk*, (or some innocent analogous sugar,) and contained no morphia nor any mineral matter! The third weighed 2 grains: it was composed of calomel and morphia, the latter forming about one-fourth the weight, or half a grain. The cause of the alarming symptoms was at once explained. It will be perceived from the analysis, that the powders differed from each other in weight by more than one half, and that the first contained a dose of morphia which might have proved fatal had it been taken according to the written order; while the second was an innocent powder, of which a person might have swallowed an ounce with impunity, and yet it was less than half the weight of the really poisonous powder! The third contained a full dose of morphia. The quantity of morphia which the patient took cannot be conjectured, as the doses of morphia in the remaining powder followed no regular rule: but it appears to have been sufficient to destroy her confidence for ever in infinitestimal doses! This case, while it shows in an unexpected way the gross deception practised on the public, and the risk of life which must be incurred by such a reckless mode of prescribing, affords a hint of some value to the medical jurist. Let us imagine that the patient had taken the powder containing the grain of morphia and had died from its effects, and that only the second of the powders, with the written directions, had been forwarded to a chemist for analysis. This would have been found to be sugar of milk, a substance which could be productive of no mischief. Had a charge of manslaughter even arisen, there would have been an admirable ground of defence in the allegation that the unfortunate deceased had died of apoplexy, for it would have been argued,—she could have taken nothing but sugar of milk, with, perhaps, a harmless decillionth dose of some homœpathic medicine! The prescriber under these circumstances, would probably have been triumphantly acquitted.

It is impossible to rely upon the results of experiments performed on animals with morphia and its salts, as a mode of determining their relative effects on man. Dumas justly observes that animals resist doses which would infallibly destroy a human being. (Traité de Chimie, v. 778.)

On the trial of *Castaign*, M. Chevallier, who sold the acetate of morphia to the prisoner, stated that he tried the effects of the salt upon himself. He took a quantity that had been administered to a cat and dog without injurious consequences, and he was ill for a fortnight. On the same occasion, M. Segalas declared that eight grains of the acetate of morphia might prove fatal, but in several experiments *fourteen grains* had been administered to animals without killing them. According to Dr. Vassal, the acetate has been given to horses in drachm-doses without injury; and the same writer relates the following experiments on the effects of the acetate of morphia performed on dogs by M. Dublanc. A solution of twenty-grains of acetate of morphia was injected into the stomach of a full-sized dog. There was general depression, somnolency, salivation, nausea, contractility of the pupils, retention of urine, constipation, loss of appetite, with partial paralysis of the posterior extremities. Two days afterwards forty-eight grains were injected, and as the animal resisted this dose, a drachm was given on the following day. Nineteen hours afterwards, the dog appeared in its usual health and vigour. It was then destroyed, but no particular morbid appearances were discovered. (Considerations Medico-Chimiques sur l'Acetate de Morphine, p. 92.) These results have been more recently confirmed by the experiments of Flandin on dogs, rabbits, birds, and an ape. This gentleman thinks that these extraordinary facts are to be explained by supposing that the morphia is digested and entirely decomposed by the gastric juice. (Académie des Sciences, 26 Juillet, 1847; Gaz. Méd. 31 Juillet, p. 619.) Whatever explanation may be offered, the results fully bear out the opinion elsewhere expressed, (p. 137, ante,) that experiments on animals, when the object is to determine the fatal doses of poisons, may lead to the most dangerous fallacies!

Although no difference is commonly made in the medicinal dose of morphia, or either of the salts mentioned, it may be proper to state the relative proportion of the alkaloid contained in the two latter preparations.

	Acetate.	Muriate.
Morphia	84·8	88·7
Acetic Acid	15·2	11·3
	100·	100·

The acetate is by no means a stable compound. It spontaneously loses acetic acid, even in the crystallized state,—whereby it becomes less soluble in water, and less energetic in its operation. This is considered by some to afford a sufficient explanation of the variability of its effects in given doses.

Absorption.—There is no doubt that the active principle of opium—morphia, is conveyed into the blood by absorption, with greater or less rapidity according to those circumstances which influence the process. In one instance, already quoted, there was clear proof that the muriate of morphia had been absorbed in less than *half an hour* (ante, page 493.) The odour of opium, it is well known, soon disappears, as it has not been detected in the contents of the stomachs of persons who have died in a few hours from comparatively large doses. The active principle, morphia, also disappears, but not in all cases so rapidly as to prevent the discovery of traces of it after the lapse of many hours. In a case of poisoning with ten grains of muriate of morphia, in which the individual survived twelve hours, Dr. Traill detected the poison in the stomach. That the poison is absorbed appears to be also proved by the effect produced on leeches, applied to persons who have swallowed the drug. Dr. Christison quotes an instance where leeches were applied to a child poisoned by a strong injection of poppy-heads: these animals dropped off dead. (Op. cit. 709.) Expert chemists have sought for the poison in the blood, but unsuccessfully,—a fact which is perhaps to be ascribed to the great difficulty of separating small portions of morphia from so complex a liquid. M. Barruel states that he detected morphia in the blood and urine of a man who was poisoned by a large dose of tincture of opium; but his results have not been confirmed by the researches of other chemists. Dr. Skae lately examined the urine of a person poisoned by opium without detecting morphia, although the poison was discovered in the stomach. It is not yet known whether morphia is eliminated by the kidneys, or by any other organ. Owing to the copious perspiration often observed in this form of poisoning, and the general effect of the drug to suppress the other secretions, Mr. Sibson of Nottingham has suggested that the poison may be really conveyed away by the skin, and he has proposed that means should be adopted in the treatment, to excite the cutaneous transpiration. It would certainly be desirable to have an analysis of this secretion. (See p. 614, post.)

BLACK DROP.

This is a preparation of opium, in which the morphia is combined with acetic acid, and very little meconic acid is present. In the black drop, according to Pereira, verjuice, the juice of the wild crab, is employed as a menstruum instead of vinegar. The black drop is considered to have from three to four times the strength of the tincture of opium. The formula for this preparation will be found in Dr. Neligan's work, On Medicines, &c., p. 235. According to this, it is a compound of half a pound of opium to three pints of the expressed juice of the wild crab. It resembles the *Acetum Opii*, and has more than twice the strength of laudanum.

[The black drop of the Pharm. U. S. contains about one grain of opium in 6½ to 7 minims.—G.]

SEDATIVE SOLUTION.

This is an aqueous solution of opium with a little spirit and less meconic acid than the common tincture. (Pereira, ii. 1772.) It is considered to have three times the strength of tincture of opium, but there is so great a difference of opinion on this point, that Dr. Neligan represents it as being of only about the same strength as laudanum. (Medicines, &c., 236.) He states that it is composed of three ounces of extract of opium, six drachms of spirit, and as much distilled water as will make up two pints. It appears to be an energetic preparation. Mr. Streeter stated at the Westminster Med. Soc., Dec. 1838, that he had known one drachm and a half of it to prove fatal to a lunatic; and twenty minims of the solution destroyed the life of an old woman. A medical gentleman, lying dangerously ill from an attack of dysentery, took, by mistake, about seven drachms of Battley's Solution. Within five minutes, salt and water, with mustard, were administered, and twenty-four grains of sulphate of zinc. Vomiting ensued: the emetic was repeated, and with the same effect; the fluid evacuated at the second vomiting having the usual smell of opium. Half a drachm of ipecacuanha was afterwards given to complete the emptying of the stomach. Notwithstanding this repeated vomiting, symptoms of narcotism presented themselves speedily, with contraction of the pupils and very great drowsiness—rendering it necessary to remove the patient from bed in his very debilitated state, and keep him constantly moving, until about 9 P. M. (seventeen hours,) when vomiting came on spontaneously;—he was then put to bed, and allowed to sleep. The original disease afterwards resumed its course (complicated by an attack of gastritis,) and at length terminated favourably; but the patient had no recollection of what had occurred for twenty-four hours after the administration of the emetics; and it appeared to his medical attendants that an excited state of the mind remained for some days afterwards. (Prov. Journ., Jan. 28, 1846, 42.)

Effects of external application.—Opium or morphia and its salts have in all their combinations, the property of affecting the body through the skin. Excepting in cases of idiosyncrasy, or where a large quantity of the drug is too frequently applied to an abraded surface, they are not likely to produce fatal effects by this mode of introduction into the system. There is, however, one instance reported of a very large quantity of laudanum having acted fatally when applied in a poultice to the unbroken skin of the abdomen. (Christison, Op. cit. 723.) In general, the narcotic preparation is only applied after the skin has been removed by a blister: but the following case is sufficient to show that small doses of a salt of morphia may in this way act very energetically.

A young woman, labouring under scirrhus of the uterus, and suffering from vomiting and pain in the stomach, was ordered to apply to the epigastrium, from which the skin had been previously removed by a blister, the 1-23d part of a grain of the muriate of morphia. The same dose was repeated by the endermic process the following morning. Some time afterwards, the woman fell into a state of complete narcotism. She suffered from pain in the head, stupor, ringing in the ears, dizziness, and incoherency, a hot and dry skin, and a strong and frequent pulse. Among the symptoms was one somewhat remarkable, namely, that she saw only the half of surrounding objects;—for instance, in the case of a person standing before her, she could only see the right or left half of the body. The cerebral congestion was followed by convulsions. Venesection was performed, but this only produced a stronger attack, followed by another. A compress, soaked in vinegar, with ice, was applied to the forehead, followed by mustard poultices to the lower extremities. The symptoms gradually abated, but it was three weeks before vision and speech were perfectly restored. (Oesterreichische Medicinische Wochenschrift, April 1845.) Four-tenths of a grain of the acetate applied to a blistered surface, have been known to cause dimness of vision and delirium. The dosé commonly recommended for application endermically, is from one to two grains.

Opiate preparations introduced into a wound, or as enemata into the rectum, may also produce fatal effects. Orfila relates the case of a man who died from the effects of an injection containing thirty grains of opium. (Op. cit. ii. 225.) A child has been killed by ten grains of the sulphate of morphia, given in the form of an enema, by mistake for sulphate of quinine. (Med. Gaz. iv. 220.) Their application to the lining membrane of the nose will produce all the usual symptoms of poisoning and death. (Wibmer, Arzneimittel. *Papaver.*)

TREATMENT.

In poisoning by morphia or its salts, the same treatment is required as in poisoning by opium. (For the CHEMICAL ANALYSIS, see post, p. 501.)

OTHER ALKALOIDS IN OPIUM.

Besides morphia, opium contains three alkaloids, Codeia, Narcotina, and Thebaina,—two neutral principles, Narcein and Meconin, and one acid, the Meconic.

Codeia is found to exert a poisonous action on animals, and it has been used in France as a narcotic. It is considered to have only one-half of the strength of morphia, with which it is in general mixed. The common muriate of morphia of the shops is, according to Pereira, a compound muriate of morphia and codeia. M. Kunckel found that, when combined with acids, it lost much of its activity.

Narcotina.—The results of experiments with this alkaloid on animals are very conflicting. In the human subject it has been observed to produce headach; but when these effects have followed, it has been probably mixed with morphia. I have frequently found this mixture in specimens; and Dr. Christison states, that he has met with narcotina in morphia, a circumstance which may tend to explain the variable effects of this alkaloid in large doses.

Thebaina (Paramorphine).—According to Magendie this substance, when injected into the jugular vein of an animal, acts like strychnia, producing tetanus and death in a few minutes. Orfila found that it produced opisthotonos, but the animals recovered. (Op. cit. ii. 203.)

Narcein, Meconin, and **Meconic Acid** are inert. Experiments show that they exert no poisonous action on the animal systen.

CHAPTER XXXVI.

OPIUM CONTINUED—CHEMICAL ANALYSIS—EVIDENCE FROM ODOUR—MORPHIA—PROPERTIES OF THIS ALKALOID IN THE SOLID STATE—ACTION OF TESTS ON THE SOLUTION—OBJECTIONS TO THE TESTS—MECONIC ACID—ITS PROPERTIES—TESTS—OBJECTIONS TO THE TESTS—PROCESS FOR THE DETECTION OF OPIUM IN ORGANIC MIXTURES—OPIUM RARELY DISCOVERED—OBJECTIONS TO THE PROCESS—SUMMARY—DELICACY OF THE TESTS FOR MORPHIA—SEPARATION AND DETECTION OF MECONIC ACID IN OPIATE MIXTURES—DELICACY OF THE TEST FOR MECONIC ACID—QUANTITATIVE ANALYSIS—ANALYSIS OF ACETATE, MURIATE, AND SULPHATE OF MORPHIA, NARCOTINA, CODEIA, NARCEIN—PROPORTION OF OPIUM IN OPIATE MEDICINES.

CHEMICAL ANALYSIS.

Opium.—There are no means of detecting opium itself, either in the solid or liquid state, except by its smell and other physical properties, or by exhibiting a portion of the suspected substance to animals, and observing the effect produced. The smell is said to be peculiar, but a similar smell is possessed by lactucarium, which contains neither meconic acid nor morphia. The *odour* is a good concomitant test of the presence of the drug, whether it be in a free state, or dissolved in alcohol or water, but it is not perceptible when the solution is much diluted. I found that half a grain of powdered opium dissolved in half an ounce of water, lost its characteristic smell by a short exposure. The odour is decidedly volatile, and passes off when an opiate liquid is heated; it also escapes slowly at common temperatures. Again, it may be concealed by other odours, or the drug may undergo some change in the stomach during life which may destroy the odour. The analysis in cases of poisoning by opium, is therefore limited to the detection of morphia and meconic acid.

MORPHIA.

Morphia is known by the following properties:—1. It crystallizes in fine prisms, which are white and perfect, according to their degree of purity. 2. When heated on platina, the crystals melt, become dark-coloured, and burn like a resin with a yellow smoky flame, leaving a carbonaceous residue. If this experiment be performed in a small reduction-tube, it will be found by employing test-paper, or a mixture of arsenious acid and nitrate of silver, that ammonia is one of the products of decomposition. 3. It is scarcely soluble in cold water, requiring 1000 parts to dissolve it; it is soluble in one hundred parts of boiling water, and the hot solution has a faint alkaline reaction. By its insolubility in water it is readily known from its salts. It is not very soluble in ether, thus differing from narcotina; but it is dissolved by forty parts of cold, and rather less than this quantity of boiling alcohol. It is soluble in oils and in the caustic alkalies (potash.) 4. It is easily dissolved by a very small quantity of all diluted acids, mineral and vegetable. 5. It has a bitter taste. In order to apply the chemical tests for morphia, it should be dissolved in a few drops of a diluted acid, which may be either the acetic or muriatic. If either the muriate or the acetate be presented for analysis, it may be at once dissolved in a small quantity of boiling water.

TESTS.—The best tests for this alkaloid are the following: 1. *Nitric acid.* This, when added to a moderately strong solution of a salt of morphia, produces slowly a deep orange-red colour. If added to the crystals of morphia or its salts, deutoxide of nitrogen is evolved:—the morphia becomes entirely dissolved, and the solution acquires instantly the deep red colour above described,—becoming, however, lighter by standing. In order that the effect should follow, the solution of morphia must not be too much diluted, and the acid must be added in pretty large quantity. The colour is rendered much lighter by boiling;—therefore the test should never be added to a hot solution. 2. *Permuriate of iron* (sesquichloride, or colourless persulphate. Either of these solutions when saturated and neutralized (by a small quantity of potash if necessary,) gives an inky-blue colour in a solution of morphia. If the quantity of morphia be small, or the test have a deep red or yellow tint, the colour is greenish. The blue colour is entirely destroyed by acids,—it is also destroyed by heat: thus the iron-test should never be employed with a very acid or a very hot solution of a salt of morphia. It should be observed, that the blue given by the test in a solution of morphia, is entirely destroyed by nitric acid and replaced by the orange-red colour, so that the nitric acid will act through the iron-test, but not vice versâ. In this way two tests may be applied to one quantity of liquid. 3. *Iodic acid.* Morphia in the solid state or in solution decomposes this acid, taking part of its oxygen, and setting free iodine. In order to make this evident, the iodic acid should be first mixed with starch; and a part of this mixture only, added to the suspected solution,—part being reserved to allow of a comparison. If the iodic acid be added to the solution of morphia without starch, the liquid becomes brown and smells of iodine. If the quantity be very small, there is only a reddish or purple tint slowly produced:—if large, the dark-blue iodide of farina is formed in a few seconds. This colour being destroyed by heat, the test must not be added to a hot solution. This test succeeds equally well with morphia or its salts when unmixed with organic matter; but the analyst must remember, that the blue iodide of farina forms a colourless combination with a large quantity of starch: hence but little of this substance should be used, if the quantity of morphia be small. 4. *Sulphuric acid* and *chromate of potash.* When strong sulphuric acid is poured on pure morphia in a solid state, there is either no effect, or the alkaloid acquires a light pinkish colour. On adding to this a drop of chromate of potash, it immediately becomes green (from oxide of chrome,) and retains this colour for some time. Other alkaloids are not thus affected. Narcotina is turned of a bright yellow by sulphuric acid; therefore, although it becomes green with chromate of potash, it could not be mistaken for morphia: besides the green rapidly passes to a dingy brown colour.

Objections to the tests.—1. *Nitric acid.* This test gives an orange-red or yellow colour with gallic acid and some organic liquids; but this colour is not likely to be confounded with the deep red tint of morphia:—besides, it is presumed that there is no organic matter present. It may be mentioned that, according to some, *pure* morphia is not thus affected by nitric acid. I have tried many specimens, some of a very pure kind, but have never failed to obtain this result. A more important objection is, that other alkaloids are similarly coloured by it:—thus common *Strychnia,* from admixture with brucia, is turned of a scarlet colour; but does not readily dissolve in the acid,—small red lumps remaining in it for some time. Again, *Brucia* is turned of a red colour, but it is dissolved. In each of these cases, the red colour in the course of half an hour changes to a greenish-brown, while the orange-red of morphia becomes of a light yellow. *Narcotina* is turned at once of a bright yellow colour by nitric acid, and therefore cannot be mistaken for morphia. *Delphinia* is said to give a similar colour, but I have found that this alkaloid produces with nitric acid at first a yellow, and then an iron-rust red solution, while the undissolved portions remain of a dingy yellow colour. *Veratria* gives a light red tint. If the objections to the test be not thus removed, it may be observed that none of the above alkaloids decompose iodic acid and set free iodine. 2. *Permuriate of iron.* This gives a blue tint with all solutions containing tannin or gallic acid, but the test could not with any propriety be used in an organic mixture;—or at least, in such a case, no safe inference could be drawn from the production of a blue colour. The presence of meconic acid in combination with morphia, entirely prevents the action of this test on the alkaloid. 3. *Iodic acid.* I have found this acid to be liable to spontaneous decomposition when long kept; and thus it should be separately tested with starch, before it is added to the suspected liquid: this will show whether any free iodine be present or not. Iodic acid is similarly decomposed by sulphocyanide of potassium, sulphuretted hydrogen, cyanide of potassium, sulphurous and gallic acids and oxalic and meconic acids under exposure to heat, as well as by other bodies; but as we are now supposed to be examining a pure alkaline solid dissolved for the occasion, these objections are of no moment. Sulphocyanide of potassium gives a reddish colour with nitric acid, but it forms a deep cherry-red compound with the iron test; thus entirely differing from morphia. An important fact is, that no alkaloid yet discovered, excepting morphia, possesses the property of decomposing iodic acid. These tests taken together, therefore, establish the presence of morphia. The objections to which one is exposed, if we except gallic acid, are removed by the application of the others; but gallic acid is easily known from morphia by its ready solubility in boiling water, and the acid reaction of its solution. If the salt of morphia be presented for examination in a state of solution, it should be evaporated to crystallization, and then redissolved in a small quantity of water.

MECONIC ACID.

This is a solid crystalline acid, seen commonly in scaly crystals of a reddish colour. It is combined with morphia in opium, of which, according to Mulder, it forms on an average six per cent. (Brande, 1200;) and it serves to render that alkaloid soluble in water and other menstrua. It is dissolved by one hundred and twenty-five parts of cold water: it is much more soluble in boiling water, but is in great part precipitated on cooling. The cold saturated solution has, notwithstanding its sparing solubility, a strongly acid reaction. The solution, when very much diluted, is precipitated of a yellowish-white colour by acetate of lead (meconate of lead;) and in reference to the detection of the acid in medico-legal analysis, it is proper to observe, that the meconate of lead is insoluble in acetic acid—a property which allows it to be thus easily separated not only from some of the organic compounds of the oxide of lead, but also from the sulphocyanate of lead, which is quite soluble in acetic acid. Like all the vegetable salts of lead insoluble in water, the meconate is very easily dissolved by nitric acid. Meconic acid is precipitated white on the addition of lime-water (meconate of lime;) but this precipitate is easily dissolved by acids, even by those of the vegetable kingdom. A mineral salt of lime (chloride of calcium) produces no precipitate in a cold saturated solution of meconic acid. These results appear to me to show that a salt of lead is preferable to a salt of lime as a precipitant of meconic acid. The acetate of lead is commonly used for this purpose in organic mixtures suspected to contain meconate of morphia; but for reasons above given, the liquid should always be slightly acidulated with acetic acid before the salt of lead is added.

TESTS.—Many tests have been proposed for meconic acid; there is only one upon which any reliance can be placed, namely, the *Permuriate* or *Persulphate of iron*. This test gives, even in a very diluted solution of meconic acid, a deep red colour;—and it is owing to the presence of this acid, that the salt of iron causes a red colour in the tincture or infusion of opium, as well as in all liquids containing traces of meconate of morphia,—the effect of the iron test with morphia being counteracted by the presence of meconic acid. The red colour of the meconate of iron is not easily destroyed by diluted mineral acids, by a solution of corrosive sublimate, or by chloride of gold, but it is by sulphurous acid and chloride of tin.

Objections to the test.—It has been objected to the application of the iron-test, that *Sulphocyanic acid* or an alkaline *sulphocyanide* produces a similar colour with a per-salt of iron. This is certainly the case, but the red colour produced by the sulphocyanide is immediately destroyed by a few drops of chloride of gold or bichloride of mercury. The liquid may be diluted, and a few drops of a solution of acetate of lead added;—a precipitate falls, either as meconate or sulphocyanate of lead. The former is insoluble, while the latter is quite soluble in acetic acid. It has been recommended by Dr. Percy to adopt another method for distinguishing the meconic from sulphocyanic acid. The red liquid should be acidulated with diluted sulphuric acid, and pure zinc dropped into it. If the red colour be due to the sulphocyanate of iron, it will disappear, and sulphuretted hydrogen gas will be evolved, known by its odour, and its reaction on paper dipped in a salt of lead and plunged into the mouth of the tube. Dr. Christison describes this as the best distinction yet proposed. (Op. cit. 690.) I have found it liable to a dangerous fallacy, which led to a mixture containing meconic acid being pronounced to be sulphocyanic acid by a gentleman well versed in chemical analysis. He had used for the purpose of the experiment distilled zinc, in as pure a state as he could possibly procure it from a chemist's. This, on examination, was found to contain sulphur, which at once explained the cause of the mistake. Nine different specimens of zinc, procured from different places, were tried, but all contained sulphur: hence, in resorting to this mode of distinction, it is indispensable that the analyst should first test his zinc, to be quite certain that no sulphur is present. From the difficulty of procuring absolutely pure zinc, it will be found more convenient, and quite as satisfactory, to employ a solution of bichloride of mercury as the means of making a distinction between the meconate and sulphocyanate of iron. *Indigotic acid.* Mr. Cooper states that some of the acids of indigo redden the persalts of iron like meconic acid, and recommends as a distinction the use of the potassa-chloride of gold, which produces no change in these acids, while with meconic acid the mixture is blackened, as will be presently described. This cannot, however, be regarded as a practical objection to the test, because the indigotic acids are preparations of too great rarity to find their way into mixtures requiring a medico-legal examination on a suspicion of poisoning. *Strong acetic acid* or an alkaline *acetate.* Concentrated acetic acid, or any of the alkaline acetates in a concentrated solution, give a red colour with a persalt of iron, and this is not removed by the metallic chlorides just mentioned, in which respect these salts resemble a solution of meconic acid;—but as they are neutral, and on boiling with or without an acid, acetic acid is immediately evolved, no difficulty can arise on this ground. There are other objections to the employment of the iron-test for the detection of meconic acid, but as these refer to the action of the test on mixtures containing organic matter, the description of them will be for the present reserved.

The *Potassa-chloride of gold* has been proposed as a test for meconic acid. The mode of employing this test, is as follows. We dilute the acid, add two or three drops of a solution of caustic potash, and then a like quantity of a solution of terchloride of gold. There appears either immediately, or in the course of a few minutes, according to the degree of dilution, a deep purple-black cloudiness, arising from reduced gold, which slowly spreads throughout the mixture. The same effect takes place in an opiate liquid containing even a small quantity of meconic acid, unless it be too much diluted. The great objection to this test is, that a similar decomposition takes place with morphia, gallic acid, and many other deoxidizing substances; but it does not occur with the sulphocyanides.

DETECTION OF OPIUM IN ORGANIC MIXTURES.

Opium not detected.—Opium itself may be regarded as an organic solid, containing the poisonous salt which we wish to extract. It is not often that, in fatal cases of poisoning by opium or its tincture, even when these are taken in large quantity and death is speedy, that we can succeed in detecting meconate of morphia in the stomach. It is probably removed by vomiting, digestion, or absorption:—certainly not always by decomposition; for I have found meconate of morphia in organic liquids kept for twelve and fourteen months, and allowed to decompose spontaneously under the free access of air. In the case of a young woman, who died *five hours* after taking *two ounces* of laudanum, Dr. Christison did not succeed in detecting morphia by any of the tests. Other cases of a similar kind are mentioned by him. In two instances of poisoning, I have been unable to detect any meconate of morphia,—in one, the man died twenty-four hours after taking nearly two ounces of the tincture: in both of these, however, the stomach-pump had been applied. The following case occurred in June 1836. A woman swallowed an ounce and a half of laudanum in beer. In half an hour she was in a state of profound coma: she died in nine hours. None of the poison could be detected in the stomach,—there was not even the smell of opium. In two cases, which occurred in 1844, one having proved fatal in five, and the other in twenty-two hours, I could not detect the least trace of opium either by the odour or by tests. In the latter case, half an ounce of the tincture had been taken. On the other hand, Dr. Skae detected traces in one instance, where probably only two drachms of the tincture had been taken. (See ante, p. 498.) The poison may in general be detected more easily in the matter vomited during life (if vomiting should have occurred) than in the contents of the stomach after death. It was thus detected readily in the form of aqueous infusion, in a case where the contents of the stomach had been ejected about seven hours after the poison had been swallowed. (Med. Gaz. xxxvii. p. 724.)

Lassaigne's process.—If the matter be solid it should be cut into small slices;—if liquid, evaporated to an extract, and in either case digested with distilled water and a small quantity of acetic acid for one or two hours at a gentle heat. The aqueous solution should be filtered, some acetic acid added, and then acetate of lead until there is no further precipitation. The liquid should be boiled and filtered (meconate of lead is left on the filter while any morphia passes through under the form of acetate. The surplus acetate of lead contained in the solution, should now be precipitated by a current of sulphuretted hydrogen—the sulphuret of lead separated by filtration, and the liquid evaporated at a very gentle heat to an extract, so that any sulphuretted hydrogen may be entirely expelled. On treating this extract with alcohol, the acetate of morphia may be dissolved out and tested. The meconate of lead left on the filter, may be decomposed by boiling it with a small quantity of diluted sulphuric acid; and in the filtered liquid, neutralized if necessary by an alkali, the meconic acid is easily detected by the iron-test. This analysis requires care as well as some practice in the operator, in order that the morphia should be obtained in a sufficiently pure state for the application of the tests. Before resorting to this process, it is advisable to employ *trial tests*, in order to determine whether any meconic acid or morphia be present or not. The smell of opium may be entirely absent. The best trial tests are nitric acid and the permuriate of iron. These will give in the infusion or liquid, if it contain opium, the changes already indicated. In testing for meconic acid, it is advisable to dilute the organic liquid, if coloured, with a sufficient quantity of water, to render the production of a change of colour by the test, perceptible. In respect to this method of detecting the meconate of morphia in a suspected liquid, it is proper to observe, that nitric acid will indicate the presence of morphia,—and permuriate of iron the presence of meconic acid, in infusions containing so small a quantity of opium as not to be precipitated by the acetate of lead.

Objections.—The tests for morphia, when applied to organic liquids, are open to objections. Thus nitric acid as well as permuriate of iron added to a decoction of *White mustard*, or to a solution of a *Sulphocyanide*, produces effects very similar to those produced in a solution of morphia. The only difference is that the red colour of the meconate of iron is not destroyed by a solution of corrosive sublimate, while the red colour of the sulphocyanide is. Another remarkable circumstance to be observed is, that iodic acid is decomposed by both of these liquids, so that a decoction of mustard closely resembles in the effects of all the tests, a solution of morphia. But circumstances may show that the liquids could have contained no mustard,—that on evaporation it left no residue of sulphocyanide of potassium, and finally that it had the odour of opium. Dr. Pereira states that he has obtained from the stomachs of subjects in the dissecting-room (not poisoned by opium) a liquid which reddened the salts of iron. (Mat. Med. ii. 1741.) I have applied the iron-test in a large number of cases to the contents of the recent stomach just so diluted as to allow any change of colour to be perceptible, without obtaining this result. On one or two occasions a greenish-brown colour was produced which could not have been mistaken for the meconate of iron. Such an objection could moreover refer only to the application of the iron-test to the organic liquid, as a preliminary experiment for guiding the analyst; but it is impossible to rely upon a mere change of colour in an organic liquid as positive evidence of the presence of meconic acid;—such an inference would only be justifiable where the result was obtained from the decomposition of the precipitate, produced by a salt of lead in the mixture. Therefore, such liquids in order to create ambiguity must like meconic acid, be precipitated by acetate of lead, and the washed precipitate must possess all the properties of the meconate of lead. As an additional corrective when the iron-test is employed in a preliminary experiment, it must be remembered that the red colour produced by it in an *opiate* liquid, is changed to a golden red by the addition of nitric acid: the only other liquid in which I have found this to occur, was in a strong decoction of white mustard. Nitric acid produces a red colour like that of morphia with an infusion of *Nux vomica*, but the iron-test gives a green and not a red tint to this infusion. These tests may be usefully employed, with the precautions above mentioned, to determine the presence or absence of opium in the contents of the stomach of a living person, ejected by vomiting or removed by the stomach-pump.

Saliva.—On a trial which took place at Edinburgh in 1829, (*Stewart*,) Dr. Ure pronounced an opinion that opium was present from the action of the iron-test indicating the presence of meconic acid. He was unable to procure any evidence of the presence of morphia. It was objected to his evidence that the saliva, from containing sulphocyanic acid or a sulphocyanide, might produce a similar effect. From numerous experiments on the saliva, I have found the results to be rather variable. It is sometimes rendered of a bright red by the addition of a persalt of iron to about half an ounce, at other times it is scarcely affected; but the reddening effect will be found to take place more commonly than not. The practical question is, however, that we have to deal with the *contents of the stomach*, not with the pure secretion of the salivary organs: and it is by no means probable that the saliva will ever be found in the stomach in a state to create any ambiguity in the application of the persalt of iron even as a trial-test. The quantity will be small, and so mixed up with mucus and other organic matters as entirely to prevent the reaction. If any ambiguous change occurred, the fact that the red colour was due to a sulphocyanide in saliva, and not to meconic acid in opium, would be made apparent by the colour being immediately discharged on the addition of a drop of a solution of corrosive sublimate. This, for reasons already assigned (p. 503,) is far better as a method of distinction, than that proposed by Dr. Percy. Besides, as in the case of the decoction of mustard, the objection has no force, because it applies to the preliminary process, and not to the separation of meconic acid by a salt of lead, and the subsequent decomposition of the meconate of lead.

Saliva resembles a solution of morphia, inasmuch as it decomposes iodic acid and sets free iodine: it differs from it in being coagulated and not changed in colour by strong nitric acid,—also in giving a red and not a blue colour with a persalt of iron. One circumstance should always be borne in mind, namely, that objections of this kind can never apply to a medico-legal analysis in cases in which the suspected poisonous article has *not* been swallowed; and these form a large proportion of those which come before a medical practitioner.

A Decoction of Laburnum.—It has been recently announced that meconic acid is actually contained in the bark of the common laburnum tree, and that the iron-test strikes, with a decoction or infusion of this bark, a deep-red colour, characteristic of meconic acid. The writer has further asserted that in testing for meconic acid, laburnum or laudanum would give precisely similar results, so that if the stomach contained a quantity of laburnum bark the process above described would lead to an erroneous suspicion of the presence of opium. (Pharm. Times, Feb. 20, 1847, p. 429.) The improbability of laburnum bark or its decoction being found in the stomach, unless it had been intentionally administered as a poison, would be sufficient to take away the practical force of this objection, admitting it to be valid. Laburnum is a most powerful poison, and destroys life under symptoms widely different from those produced by opium (See LABURNUM;) but the result of many experiments with the concentrated and diluted decoction and infusion of the bark procured in the metropolis as well as at a distance in the country, is that I have not been able to detect in it the slightest trace of meconic acid, or of any acid that could possibly be mistaken for it. The iron-test gives at first a deep-reddish colour when added to the decoction, but this colour speedily changes to a dingy greenish-brown, instead of remaining of a clear red like the meconate of iron. It is quite certain that a person used to the analysis of opium could not mistake this chemical change for that produced by meconic acid. As tannic acid gives no precipitate with the decoction, the effect is probably due to gallic acid combined or mixed with organic matter. The clear liquid obtained from a decomposition (by sulphuric acid) of the precipitate formed in the decoction by a salt of lead, did not acquire any red colour upon the addition of the iron-test.

Summary.—It might be supposed that the chemical difficulties above described would almost suffice to justify the abandonment of the tests at present employed for the detection of meconate of morphia in opium; but, in practice, these objections will not be found to have that force which might at first view be ascribed to them. The seeds containing the elements of sulphosinapic acid are not to any extent used as articles of food: and it is extremely rare to find any traces of sulphocyanic acid in the contents of the stomach. Giving to such objections their greatest value, they would of course apply chiefly to those cases in which the contents of the stomach were under analysis; but in these cases, opium is so rarely found, that, unless death be very speedy, the search for it is almost hopeless. If, however, death had occurred rapidly, then, unless much vomiting had taken place, the poison would be found most probably, in large quantity, in the stomach, or otherwise in the vomited matters, so as to lead to its certain identification. It would be easily known, both by its chemical and physical characters; or, if these were doubtful, by the effects which the administration of some of the suspected solids or liquids, might produce on animals. In most instances a portion of the liquid swallowed by the deceased, is handed for analysis; and, with this, some account of the symptoms and post-mortem appearances may commonly be obtained. Speculative objections to the tests, on the alleged ground of the possible presence of sulphocyanic or sulphosinapic acid, would, on these occasions, be utterly inadmissible. Similar objections might be urged, with greater plausibility, to the chemical analysis of most mineral poisons; but we invariably find that such objections are quite inconsistent with the proved facts of the case.

In employing *iodic acid* as a test for morphia, it is, in general, recommended to mix the iodic acid with the solution of starch, before adding this mixture to the alkaloid or its salt. This plan may be properly pursued, in order to test the purity of the iodic acid; but so far as the testing for morphia is concerned, it will be found better to add the acid to the alkaloid or its salt, in a white saucer, when the setting free of iodine will be indicated, not merely by the liquid acquiring a brown colour, but by the easily-recognisable odour of iodine. To this a small quantity of starch may be afterwards added, when iodide of farina, either purple or blue in colour, according to the quantity of free iodine present, will result. This is preferable to employing a prepared mixture of iodic acid and starch; because, in this case, if the morphia be small in quantity, the blue iodide of farina may be dissolved and entirely concealed by the surplus quantity of starch present. In pursuing the plan recommended, the quantity of starch can be exactly adapted to the quantity of iodine set free. The iodic test cannot be safely applied to coloured liquids, but only to clear solutions which are suspected to contain morphia or one of its salts. There are many chemical objections to its employment, for which I must refer the reader to the remarks already made (p. 502.)

Delicacy of the tests for Morphia.—It has often been a matter of inquiry as to what quantity of a salt of morphia can be detected by chemical tests. (See ante, p. 502.) This question is of some interest, because it will often serve to show why chemical processes fail in detecting the poison; and thus remove a doubt which might exist in the minds of non-professional persons respecting the real cause of death in particular instances. Besides, the point at which chemical tests cease to act in detecting a poison, is a question which is often put to medical witnesses on criminal trials. It has been somewhat loosely stated that the iodic acid test will detect the 7000th part of morphia; but whether this refers to one grain of morphia dissolved in 7000 parts of water, or the 7000th part of a grain dissolved in water, is not explained, although the former is the more probable; and then it will be seen that such an experiment merely refers to the degree of *dilution* with water in which the test begins to act upon morphia, and not upon the *smallness of the quantity* of the alkaloid present. Iodic acid will, however, detect a far smaller quantity of morphia than one grain. Bearing in mind, then, the indispensible necessity for determining the effect of the test on the absolute quantity of the poison, as well as the degree of dilution employed, the following experiments were performed. The salt of morphia selected was that which is now commonly used in medicine—the muriate. Two grains of muriate of morphia were dissolved in 440 gr. (one ounce) of distilled water, and the whole accurately mixed. This formed the standard solution. 1. *Nitric acid.*—A few drops of the acid having been placed in a white saucer, the standard solution was added guttatim. With two drops, = the 110th of a grain, the liquid acquired a light yellow colour: with five drops, = the 44th of a grain, there was an orange-red tint, not perceptible, however, except in contact with a white surface. When fifteen drops of the solution, = about the 15th of a grain, of muriate of morphia were added to five drops of nitric acid in a small glass tube, there was no apparent change for two minutes; the liquid then began to acquire an orange-red tint, which was very decided when the whole was poured out into a white saucer. It speedily passed to a deep amber-red, quite characteristic of morphia. The test may act upon a still smaller quantity; but, from several trials, this appeared to be the point at which its action began to be satisfactory. Below this point, or in a larger quantity of liquid, a doubt might fairly have arisen whether the acid was acting upon a salt of morphia, or not. One circumstance requires notice: it is stated in many works on toxicology that this effect of nitric acid on morphia is known from that which it has on brucia by the red colour in the latter case being entirely discharged by the addition of a very small quantity of a solution of chloride of tin. This correcting test cannot, however, be employed under these circumstances; for the red colour produced by nitric acid on such minute quantities of morphia is so slight, that it is immediately discharged by the chloride of tin. 2. *Sesquichloride of Iron.*—One drop of the saturated solution of the iron-test was placed in a white-saucer: it had a deep yellow colour. Four drops of the standard solution of muriate of morphia, = the 55th of a grain, were then added, and the liquid acquired a green tint by no means characteristic of morphia; this green tint being probably due to the intense yellow colour of the test mixing with the blue produced by the morphia salts. If, however, the iron-test be so diluted as to destroy the yellow colour, the experiment entirely fails, even when the morphia is in comparatively large quantity. With ten drops of the standard solution, = the 22d of a grain, a blue tint began to appear, but it was by no means decided,—the quantity of water in which the salt of morphia was dissolved reduced the colour. Twenty drops of the standard solution, = the 11th of a grain, gave, in a small tube, the characteristic blue tint indicative of morphia: and it is at this point that the action of the iron-test begins to be satisfactory. When to a like quantity of the standard solution, three drops of the iron-test were added, the colour was of a dull green: showing how important it is to adjust the quantity of a test so highly coloured as this, to the quantity of morphia to be detected; and thus accounting for the frequent failures that have occurred in its employment. Further, it was ascertained, that, on adding more water to a similar quantity of the standard solution, the test ceased to act satisfactorily, the blue colour becoming lost by dilution. 3. *Iodic acid.*—Three drops of a saturated solution of iodic acid were placed in a white saucer, and one drop of the standard solution, = the 220th gr., of muriate of morphia, was added. There was immediately a perceptible odour of iodine, and the liquid acquired a faint brown tint, which passed to a light lilac colour (rapidly disappearing) on adding a small quantity of solution of starch. With two drops of the standard solution, = the 110th gr., the effect was more decided, but the colour given, on the addition of starch, was still red: no blue iodide was formed, and it was found that the red colour soon disappeared on the addition of more starch. Thus, then, the extreme limit to the action of this test, is the 220th gr., dissolved in the smallest possible quantity of water. The iodine set free by this small quantity of morphia, is perhaps better detected by its peculiar *odour* than by its action on starch. The following modification of this test may be adopted:—Place one drop of iodic acid on bibulous paper, saturated with a solution of starch, and dried: on this, place the liquid containing the fractional quantity of the salt of morphia; and the change of colour produced by the evolved iodine on the starch, is immediately rendered apparent. This test will not give satisfactory results when the liquid is much diluted or much starch is present; but it will act readily upon about the 100th gr. of a salt of morphia, if there be no unnecessary dilution of the liquid. The results, then, may be stated as follows:—Taking the first column to represent the absolute quantity of the salt of morphia; the second, the quantity of opium to which that quantity of muriate of morphia is equivalent, on the assumption that the common specimens of opium yield about five per cent. of morphia; the third, the weight of water or liquid compared with the absolute weight of the salt of morphia tested; and the fourth, the actual quantity of water or liquid employed in the experiment:—

	1. *Mur. Morphia.*	2. *Opium.*	3. *Dilution.*	4. *Water.*
Nitric Acid............	the 15 gr.......	(=1·2 gr.).........	300.........	20 drops
Sesquichlor. Iron,..	the 11 gr.......	(=1·6 gr.).........	231	21 "
Iodic Acid..............	the 100 gr.......	(=0·18 gr.).........	1300........	13 "

There is no doubt from these experiments, that iodic acid is the most delicate test for morphia; but it is at the same time open to the greatest number of objections, all of which must be removed before any inference can be drawn from its employment. The above table will serve to explain why, in the cases of young children, we can so seldom procure any evidence of the presence of opium from an examination of the liquids of the stomach, although death has taken place speedily. The quantity which has destroyed life may be actually smaller than the tests at present known will allow us to discover, even if we could succeed in separating the morphia so as to procure it in a form proper for testing. Admitting, as it is there shown, that the iodic acid will detect the morphia contained in less than the fifth part of a grain of opium, we must remember that this test cannot be safely used in coloured organic liquids containing small quantities of the drug; and that the delicacy of its reaction rather applies to the salts of morphia in a pure state, than to mixtures containing opium.

Meconic acid.—It has often been a question respecting the smallest quantity of meconic acid which should be present in a liquid, in order to admit of its separation by acetate of lead, and subsequent identification by its appropriate test—the sesquichloride of iron. This is an important point, because it substantially involves the question of the quantity of opium which should be present, in order to admit of our obtaining, in a separate state, morphia and meconic acid. A standard solution of meconic acid having been made, it was found, that when one drop containing the 1-220th grain of meconic acid was added to ten drops of a saturated solution of acetate of lead, diffused in one fluid-ounce of water, there was a visible opacity; but no perceptible quantity of meconate of lead was precipitated so long as the proportion of meconic acid was less than the *forty-eighth part of a grain.* Admitting that opium on an average contains six per cent. of meconic acid, according to the late analysis of Smyrna opium by Mulder, this is equivalent to one grain of the acid in 16·6 gr. of opium; and the forty-eighth part of a grain of meconic acid (=·0208 gr.) would be therefore represented by 0·34 gr. of common opium. Hence less than that quantity of opium, diffused in a fluid-ounce of water, would not yield with acetate of lead, a sensible quantity of meconate; and therefore the attempt to separate morphia and meconic acid by this process would be fruitless; for this cannot be done with so small a quantity, even when the pure acid is dissolved in distilled water, and is in the most favourable condition for separation. Allowing that the quantity of meconic acid contained in some kinds of opium, is greater than is here assumed—and it is said to vary from about four to nearly eight per cent.—still it is obvious, from these results, that unless the soluble matter of *several grains of opium* exists in the liquid for analysis, it will be difficult to obtain meconic acid and morphia separately. This fact sufficiently explains why opium is rarely found in the stomachs of young children who have been speedily killed by small doses, and therefore under circumstances the most favourable for the detection of its constituents. The precipitate formed in an opiate infusion by acetate of lead, is a mixture of meconate of lead with organic compounds of oxide of lead. The precipitate may appear to be copious, when the proportion of meconate is really small. Dr. Ure obtained, in one experiment, twenty-seven grains of impure meconate of lead from 100 grains of opium; but the precipitate must have contained much impurity, probably one-half of its weight. Not more than eight per cent. of meconic acid were found by Mulder in examining five specimens of Smyrna opium.

It may be readily supposed that dilution has some influence on the precipitation of meconic acid by the acetate of lead. When the solution of acetate of lead was mixed with the forty-eighth part of a grain of meconic acid, dissolved in a few drops of water contained in a small tube, there was a speedy subsidence of the precipitated meconate of lead, and it was collected within a small compass. When, however, the precipitation took place in the midst of a fluid-ounce of water, the precipitate was spread in a finely pulverulent state over a large surface of glass, whence it was difficult to detach it for subsequent analysis. This shows that it is advisable, when operating on small quantities, to have the liquids in as concentrated a state as possible.

Another question is, *What quantity of meconate of lead is required in order to yield sufficient meconic acid for the determination of its presence by the iron-test?* There are various ways in which meconic acid may be separated from the meconate of lead for this purpose. Dr. Hare originally proposed the digestion of the precipitate with sulphuric acid; and this, while it is the more speedy way of demonstrating the presence of the acid, is the only plan applicable to its detection when the quantity of meconate on which we have to operate is very minute. One-half grain of meconate of lead, equivalent to about one-fourth of a grain of meconic acid, was digested in a watch glass with a few drops of diluted sulphuric acid; and the sulphate of lead being allowed to subside, one or two drops of the sesquichloride of iron were added to the supernatant liquid, when the red colour, indicative of meconate of iron, was immediately produced. From the intensity of colour acquired by the solution, there was no doubt that even so small a quantity as one-eighth of a grain of meconate of lead, equivalent to about one-sixteenth of a grain of meconic acid, would, when properly treated, allow of the separation and detection of this body. But the meconic acid may be detected in a quantity of meconate of lead which it would be very difficult to collect in a solid form; as, for example, the sixteenth of a grain. Thus, one forty-eighth part of a grain, dissolved in a small quantity of water, was precipitated by a few drops of a saturated solution of acetate of lead in a narrow tube. When the precipitate had subsided, the clear supernatant liquid was poured off, and two or three drops of concentrated suphuric acid were added to the moist meconate of lead still contained within the tube. A small quantity of water was added, and the heat of combination sufficed, without boiling, to decompose the meconate; the meconic acid rising into and becoming dissolved by the clear supernatant liquid. The iron-test added to this gave the strongest indications of meconic acid. When the experiment was performed with a large quantity of water, the effect was rendered obscure by dilution.

It sometimes happens, in decomposing impure meconate of lead by boiling it with diluted sulphuric acid, that the liquid acquires a deep-red tint, which may interfere with the action of the iron-test. It was found, on carefully evaporating this liquid, which is a mixture of sulphuric acid, meconic acid, and colouring matter, that the latter became carbonized; and that from the evaporated residue a clear aqueous solution was procured, which readily admitted of the use of the iron-test without any risk of fallacy. This experiment shows that diluted sulphuric acid does not entirely transform meconic into komenic acid, as it is alleged in some chemical works.

There is no doubt that Dr. Hare's plan of decomposing the meconate of lead by sulphuric acid is the best, provided we take care to avoid the use of too much acid. It has been recommended to employ a current of sulphuretted hydrogen gas for the same purpose, the precipitate being diffused through a large quantity of distilled water. This answers very well when the proportion of meconate is large; otherwise it is inapplicable: and as it requires a much longer time for the completion of the experiment than when sulphuric acid is used, there does not seem to be any good reason for abandoning the use of the acid for that of the gas. The following plan may be occasionally useful: it appears to be in some respects an improvement on the use of sulphuretted hydrogen, as it is much more expeditious, and equally satisfactory in its results. A few grains of impure meconate of lead, obtained by the precipitation of an opiate infusion, may be rubbed with hydrosulphuret of ammonia diluted with water, and filtered. The liquid is boiled until acetate of lead gives no brown precipitate with it; meconic acid is then easily detected in it by the iron-test, under the form of meconate of ammonia. If too much hydrosulphuret be used, the liquid will retain a yellow colour which it only loses after long boiling,—the sulphur becoming thereby precipitated as a pale yellow sediment. This process requires more meconate of lead for its successful employment than that by sulphuric acid.

Delicacy of the Test for Meconic Acid.—A standard solution was made of two grains of the crystalline acid in 440 grains of water, allowed to become equally diffused. A drop of sesquichloride of iron, diluted with two drops of distilled water to remove the yellow colour, was placed in a white saucer, and one drop of the standard solution (= the 220th grain of meconic acid) was added: it gave instantly an intensely red colour, which was not discharged by adding to it an equal bulk of a saturated solution of bichloride of mercury. The colour was as intense as that produced by the addition of the iron-salt to a few drops of a strong solution of sulphocyanide of potassium; and the difference between the red meconate and red sulphocyanate of iron was immediately apparent on adding to the latter a like quantity of bichloride of mercury, when the colour was instantly destroyed. The standard solution was now so diluted, that each drop contained less than the 13,000th of a grain of meconic acid. The iron-test began to act with five drops of this liquid (=the 2,640th of a grain.) With fifteen drops (= the 880th grain,) a clear red was brought out, best seen in a tube on the iron-test first reaching the upper stratum of the acid liquid, but becoming somewhat faint by agitation. With twenty-five drops (= the 570th grain,) there was a decided red colour, characteristic of the meconate of iron, which was further confirmed by the colour not being discharged on adding five drops of a saturated solution of bichloride of mercury. This is the point at which the test begins to act for medico-legal purposes, provided we operate with a minimum of water, as dilution renders the result obscure. With thirty-five drops (= the 377th grain) the action of the test was of course much more strongly marked.

It will be seen from these results, that the test for meconic acid is far more sensitive than any of the tests for morphia; 2, that its action is subject to less interference from the presence of organic and other matters; and 3, that these properties render it well adapted as a trial-test for opium in all mixtures the nature of which is unknown. The 50th of a grain, or the smallest visible quantity of solid meconic acid, is easily detected by the iron-test when free: but here we see that, in solution, provided the quantity of menstruum be small, less than the 500th part of a grain may be readily discovered. Thus we may obtain the evidence of the presence of meconic acid in a liquid from a quantity far smaller than would be sufficient to furnish a separable precipitate of meconate of lead by the addition of a solution of the acetate. The preceding experiments show that while it requires at least *one-third of a grain of opium* to give a precipitate of meconate of lead in a minimum of water, the meconic acid of less than the one-hundredth of a grain of opium may be detected by the direct application of the iron-test. The use of the acetate of lead merely enables us to concentrate the meconic acid,—to collect it in a solid form and in a small bulk. The following table of results is intended to show, in the first column, how small a fractional quantity of meconic acid may be detected; in the second, the quantity of opium, in decimals, to which it corresponds; in the third, the proportion of liquid, by weight, in which the meconic acid was dissolved; and in the fourth, the actual quantity of liquid present.

1. *Meconic Acid.*	2. *Opium.*	3. *Dilution.*	4. *Water.*
The 220th gr.	(=·075 gr.)	880	4 drops.
The 377th gr.	(=·043 gr.)	13,572	35 "
The 570th gr.	(=·028 gr.)	14,820	25 "
The 880th gr.	(=·018 gr.)	14,080	16 "
The 2,640th gr.	(=·006 gr.)	15,840	6 "

It is apparent from this table that the meconic acid contained in less than the one hundred and sixtieth part of a grain of opium, may, under favourable circumstances, be made visible by the application of the iron-test. This explains why meconic acid may be often detected in an analysis, when morphia cannot;—a fact which has been repeatedly, noticed by medical jurists. It also shows, that, when the iron-test, *cæteris paribus*, gives no red colour in an unknown liquid, we may safely say that the quantity of opium, if any be present, is too small to admit of detection.

It will be understood, that in these experiments on the smallest quantity of *opium* susceptible of detection by tests, the inferences are derived from the use of *pure* meconic acid and a *pure* salt of morphia in very small quantities of water. The analyst must make due allowance for the meconate of morphia being mixed up with other principles in opium, as well as for the concealment or modification of the colours produced by the tests when the opium is diffused in a large quantity of water or in viscid alimentary matter. In such cases, before the tests are employed even for a preliminary trial, the liquid should be evaporated to an extract, and a fresh infusion or decoction made from the residue, by means of water mixed with a small quantity of alcohol.

In some experiments with *powdered opium*, it was found that the soluble part of one-tenth of a grain in one drachm of water, was clearly affected by nitric acid; but when mixed with one ounce of water it was not. In the last case, however, meconic acid was distinctly indicated by the action of a persalt of iron; but the tenth of a grain of opium in an ounce of water gave with acetate of lead, no precipitate of meconate of lead, and the same quantity in one drachm of water, gave only a faint precipitate which would scarcely admit of being collected. The meconic acid may be easily determined to be present by the iron-test, in a liquid containing only one grain of opium: but it would be difficult in most cases to obtain so small a quantity of precipitate as this would yield, in a state to allow of the separation of the meconic acid. So nitric acid might indicate morphia, when the quantity of opium in a liquid amounted to one grain or less, in which case the proportion of morphia might vary from one-fourteenth to one-twentieth part of a grain. This would not admit of easy separation: hence unless we obtain a tolerably free precipitate of meconate of lead, insoluble in acetic acid, it will not be in our power to obtain the morphia and meconic acid separately. On the whole, it is obvious that the tests for opium are far inferior in delicacy and certainty to those employed for arsenic and other mineral poisons.

Dublanc's process.—In January 1824, M. Dublanc announced to the Institute, a method for detecting morphia, or its poisonous salts, in opiate mixtures. Vauquelin reported favourably of it. It depends on the principle, that morphia forms a very insoluble compound with tannin or tannic acid. A perfect solution of the suspected matter is obtained in water acidulated with acetic acid, and this is boiled to coagulate any albumen. It is then concentrated, and fresh-made tincture of galls is added to it. Tannate of morphia is precipitated with probably some organic combinations of tannic acid. The precipitate is collected on a filter, and washed with a small quantity of water. It is then digested in pure alcohol, in which the tannate of morphia is quite soluble, while the other tannates remain behind, and acquire greater firmness and consistency by the action of the alcohol. To the alcoholic solution of the tannate of morphia, a solution of gelatin is added: this precipitates the tannin, and leaves the morphia dissolved in alcohol, from which it may be obtained by crystallization. In this way, Dublanc demonstrated the presence of one grain of acetate of morphia added to fourteen ounces of blood.

Although this process was published in France nearly a quarter of a century ago, (Considerations Médico-Chimiques sur l'Acetate de Morphine, par le Docteur Vassal, et Dublanc Jeune, Paris, 1824, p. 87,) it has been brought forward in two respectable French journals, as a recent discovery of a M. Merum!

(Journal de Chimie Médicale; Gazette Médicale, 24 Avril, 1847, 326.) The only suggestion made by the new claimant is, that the aqueous extract obtained with acetic acid, should be digested in alcohol, to free it of some organic principles before the addition of tincture of galls. I doubt whether it is so delicate as the process already described for the detection of morphia; and it is wholly unfitted for the demonstration of the presence of that important substance,—meconic acid.

Flandin's process.—On the 26th of July, 1847, M. Flandin read before the Academy of Sciences, a memoir respecting an improved method of detecting morphia in poisoning by opium. His process is based on two facts—1, that morphia as well as other alkaloids undergoes no decomposition, in contact with animal matter, at and above 212°; and 2, that ammonia precipitates them from their solutions in very minute proportions. The substances suspected to contain the alkaloid, should be thoroughly dried in a sand-bath, at a temperature not exceeding 239°. They are then to be broken up, and reduced to a very fine powder. This powder is to be digested in cold water acidulated with from 1-20th to 1-40th part of acetic acid: or in place of this, we may employ alcohol, to which a very small portion of oxalic or tartaric acid has been added. The object of using the acid liquid, is to render the vegetable alkaloid exceedingly soluble by converting it to a supersalt. The liquid is evaporated, and the vegetable salt is then procured from the residue by digestion in *cold* water. The cold aqueous solution is precipitated by ammonia, and the alkaloid is thus procured in a pure and crystalline condition. In the analysis of the urine or of syrupy liquids, they should be evaporated to an extract, and thoroughly dried by the addition of pure alumina in powder. This powder may then be digested in cold water acidulated with acetic acid as above described, and the morphia precipitated by ammonia. (Gaz. Méd. 31 Juillet, 1847.) M. Flandin describes this process as exceedingly satisfactory: but additional experiments are required to determine how far it is generally applicable. The meconic acid must be sought for by other methods.

Morphia in the tissues, blood, and secretions.—If we except the recently announced results obtained by M. Flandin, it is questionable, notwithstanding the physiological proofs of the absorption of this poison, whether it has ever been unequivocally detected in the substanse of the viscera, the blood, or secretions. A small quantity entering into the circulation is sufficient to destroy life—the diffusion of this through the mass of blood would render it impossible to determine its presence even in ten or twelve ounces of the fluid, by processes which are admitted to be far inferior in delicacy to those used for mineral poison. In a series of experiments made in 1824, M. Dublanc mixed one grain of acetate of morphia with fourteen ounces of blood, and demonstrated the presence of the alkaloid by a process just now described, to the satisfaction of MM. Vauquelin and Pelletier, who were appointed to inquire into the subject by the French Academy. (Vassal, Considérations Médico-Chimiques, &c. p. 87.) The test which he employed was nitric acid. He failed in showing the presence of the salt in an equal quantity of urine, and he entirely failed in detecting morphia in an absorbed state, *i. e.* in the bodies of persons or animals poisoned by it. Dr. Vassal thinks, and it is not improbable, that morphia, on becoming absorbed, may undergo some chemical or physical change, so as to be no longer separable from the blood, secretions, or tissues, in a crystallizable state. (Op. cit. 97.) This opinion is partly confirmed by the recent observations of Flandin. According to this experimentalist, the poisonous salt of morphia becomes changed and neutralized in contact with the living animal fluids: but the whole is not thus transformed or lost. According to the dose, traces of it may be still found in the alimentary canal, or in the circulation. He states that he has thus detected it in the fæces, urine, and the tissues of the soft organs; and he holds the opinion that that portion of the poison which kills, remains as such in the organs, and that it is in the power of chemistry to detect it. (See his process, supra.) Admitting that M. Flandin's views are confirmed by the researches of others, it is obvious that the conditions required for the detection of this poison in the tissues or liquids, can rarely exist. When given in small doses and it proves fatal, it appears to me that it will be in vain to seek for it by any chemical processes at present known. Thus, then, it is only when so much of the poison has been taken, as was more than sufficient to destroy life, and a portion of this surplus quantity remains in the stomach or intestines, that the analyst can have any hope of detecting it. Even here, great difficulties attend the research (ante, p. 505.) Dr. Vassal believes from his observations, that absorption of the poisonous alkaloid goes on for a certain period after death. He therefore recommends that the inspection should be made, and the contents removed, within ten or twelve hours from the time of death.

QUANTITATIVE ANALYSIS.—There are no satisfactory means of determining the *quantity of opium* present in a suspected liquid. Dr. Ure has recommended that we should rely upon the depth of colour produced, on the addition of sesquichloride of iron to the liquid,—considering that the intensity of the red colour will indicate the presence of a large quantity of meconic acid, and consequently of meconate of morphia. The late analyses of Mulder have shown that this method is not accurate. The meconic acid does not bear any constant proportion to the morphia in opium. The only plan is to extract the morphia and weigh it.

ACETATE OF MORPHIA.—This salt is seen in fine prismatic crystals. It melts and burns when heated. It is soluble in water, but if, as is very commonly the case after it has been made some time, it has lost any of its acid, it becomes less soluble. A few drops of acetic acid, however, will immediately restore its solubility. It gives all the reactions with the tests for morphia. (See ante, page 501.) The proportion of morphia contained in this salt is elsewhere given (p. 497.) When wetted and warmed, there is an odour of acetic acid: this acid is detected by boiling the salt with a small quantity of diluted sulphuric acid.

MURIATE OF MORPHIA.—This salt crystallizes in fine prisms, which are permanent in the air, but it is most usually seen in the form of a fine white powder. Heat produces on it the same effect as on morphia. It is soluble in from sixteen to twenty times its weight of water, forming a neutral solution. It is much more soluble in boiling water: a hot saturated solution cooled to 60° retain seven parts per cent. (Christison.) The medical solution contains only one per cent. Like the acetate and all the preparations of morphia, it is very soluble in alcohol. It contains rather more morphia weight for weight than the ACETATE, but is more certain in its composition. In order to discover the alkaloid, the tests already described may be employed (ante, page 501.) For the detection of the acid, we may add to the aqueous solution, nitrate of silver and nitric acid. (See Tests for MURIATIC ACID, p. 195, ante.)

The SULPHATE OF MORPHIA is a remarkably soluble salt, being taken up by two parts of water. According to Liebig it contains only 75 per cent. of the alkaloid. The acid of this salt may be detected by the nitrate of barytes. (See SULPHURIC ACID, page 175, ante.) There is a NITRATE OF MORPHIA obtained in a crystalline state by means of very diluted nitric acid. It is soluble in two parts of water.

It may be observed of all these salts, that they have an intensely *bitter taste:* that the morphia is precipitated from their solution, if moderately concentrated, by ammonia, but not readily by caustic potash or soda. The solution is also precipitated, even although much diluted, by tannin or tannic acid, but not by gallic acid.

In *Organic mixtures* they could only be separated by digesting the extract with alcohol, in which they are all very soluble. They are not very soluble in ether; hence the alcoholic extract might be freed from other impurities by this menstruum.

Narcotina.—This alkaloid is of but little importance to the medical jurist, but it is proper to state the chemical differences which exist between it and morphia. Its crystals have a bright nacreous lustre. It is not very soluble in water, but it is easily dissolved by boiling alcohol and ether. Unlike morphia, it is not very soluble in caustic potash or diluted acetic acid, even on boiling. When nitric acid is poured on the crystals, they acquire a yellow colour, not a golden-red, like morphia. Sulphuric acid gives to narcotina a bright sulphur-yellow colour; to morphia a pinkish-brown tint. If to the mixture of acid and alkaloid, a drop of a solution of chromate of potash be added, green oxide of chrome is set free in both cases: and if to either mixture a grain of an alkaline nitrate be then added, a deep blood-red colour is brought out,—almost black in the case of morphia (see ante, page 189.) Narcotina does not decompose iodic acid, or set iodine free. If narcotina be boiled with acetic acid, and a strong solution of hypochlorite of lime be added, the liquid acquires at first a yellow and then a red colour: morphia under the same circumstances gives a yellow liquid. This mode of testing, lately proposed by Flandin, is not very satisfactory, as the results depend much on the proportions added. When heated on platina, narcotina, like morphia, melts and burns.

Codeia.—This alkaloid, which is not often seen so well crystallized as morphia and narcotina, is known from both by its ready solubility in water, and by its forming a strongly alkaline solution. One hundred parts of water at 60° dissolve one part and a quarter; at 212° nearly six parts. It is also soluble in alcohol, and combines with acids. It differs from morphia in not decomposing iodic acid, and in not giving any red colour with nitric acid, either as a solid or when dissolved in acids. It differs from narcotina in not being turned yellow but of a light pinkish-brown colour, by sulphuric acid; but it resembles both morphia and narcotina in producing green oxide of chrome when a drop of solution of chromate of potash is added to the mixture. When codeia is boiled with acetic acid and hypochlorite of lime, it produces a yellow colour like morphia. Heated on platina it melts, forming a globule of colourless liquid: this soon darkens, and gives off a vapour which burns with a yellow smoky flame.

Narcein.—Narcein is met with in fine prismatic crystals. It is very insoluble in water, hot or cold: the hot solution has a faint alkaline reaction. It is easily dissolved by acetic acid and by caustic potash. Strong sulphuric acid turns the crystals of a blackish-green colour. If the acid be diluted with a small quantity of water, the liquid acquires slowly a light blue colour. On boiling with more water, it becomes colourless, but, by evaporation, it acquires a pinkish-red hue. Strong nitric acid dissolves the crystals, forming a light yellow solution.

PROPORTION OF OPIUM IN OPIATE PREPARATIONS.

It is necessary to state the medicinal doses and strength of some opiate preparations which are frequently used medicinally. **Confection of opium.** (*Confect. opii.*)—Contains one grain of opium in thirty-six grains, [U. S. and London; one in twenty-five grains, Dublin; one in forty-three, Edinburgh.—G.] The dose for an adult is from ten to thirty grains. **Compound soap pill.**

(*Pil. Sap. Comp.*)—Five grains contain one grain of opium. Dose three to ten grains. COMPOUND PILLS OF STORAX. (*Pil. Styracis Comp.*) The strength and dose are the same as in the compound soap pill. COMPOUND CHALK POWDER WITH OPIUM. (*Pulv. Cretæ Comp. cum Opio.*)—Forty grains contain one of opium, [London and Dublin; thirty-seven, Edinburgh.—G] Dose five to thirty grains. COMPOUND POWDER OF KINO. (*Pulv. Kino. Comp.*)—Twenty grains contain one of opium. Dose five to twenty grains. EXTRACT OF OPIUM. (*Extractum Opii.*)—Dose one quarter of a grain to three or four grains. WINE OF OPIUM. (*Vinum Opii*, or *Laudanum Liquidum Sydenhami.*)—This is said to have the same strength as the tincture, *i. e.*, one grain of opium in nineteen (twenty) drops. [The wine of the U. S. P. is stronger than that of the British Colleges, as it is a saturated vinous tincture.—G.] Dose ten drops to one drachm. External applications.—LINIMENT OF OPIUM. (*Linimentum Opii.*)—This preparation contains one drachm of the tincture in half an ounce. ENEMA OF OPIUM. (*Enema Opii.*)—In four ounces there are thirty drops of the tincture.

CHAPTER XXXVII.

PRUSSIC ACID—VARIOUS KINDS—DIFFERENCE IN STRENGTH—ACTION OF THE VAPOUR—CHRONIC POISONING—TASTE AND ODOUR OF THE POISON—CONDITIONS UNDER WHICH THE ODOUR MAY AND MAY NOT BE DETECTED—SYMPTOMS PRODUCED BY SMALL AND LARGE DOSES—ANOMALOUS CHARACTERS—INFLUENCE OF DILUTION—ITS EFFECTS CONTRASTED WITH THOSE OF OPIUM—DEATH FROM EXTERNAL APPLICATION—ACCUMULATIVE POWER—PERIOD AT WHICH THE SYMPTOMS COMMENCE—POWER OF VOLITION AND LOCOMOTION—CASES—SUMMARY OF POST-MORTEM APPEARANCES—CASES OF THE PARISIAN EPILEPTICS—QUANTITY REQUIRED TO DESTROY LIFE—FATAL DOSE—RECOVERY FROM LARGE DOSES—PERIOD AT WHICH DEATH TAKES PLACE—TREATMENT.

General Remarks.—HYDROCYANIC or PRUSSIC ACID, owing to its rapid and unerring effects when taken even in comparatively small doses, is one of the most formidable poisons with which we are acquainted. Most toxicologists consider it to be a narcotic poison, and in deference to this general opinion, I have still placed it under the section of narcotics: but from what will be hereafter stated, there is perhaps some reason to regard it as a narcotico-irritant. Its operation, as a sedative or narcotic, is, however, in general so rapid that its irritant effects are not manifested. The pure or anhydrous acid requires no notice here; since it is not likely to be met with out of a chemical laboratory. The common acid is a mixture of this pure acid with water, and sometimes with alcohol. As it is sold in shops, it varies considerably in strength. I have found different specimens to contain from 1·3 to 6·5 per cent. of the strong acid; but two varieties are now commonly met with—1. The Prussic acid of the London Pharmacopœia, containing about two per cent. (Phillips.) 2. Scheele's acid, containing from four to five per cent. In a case of poisoning which I had to investigate in July, 1847, the acid which was sold for Scheele's was found to contain only *two* per cent.! (Med. Gaz. xl. 171.) In another instance there was the same deficiency of strength. In short, there is no certainty respecting the strength of any two specimens sold as Scheele's acid, a subject which requires the very serious consideration of medical practitioners who prescribe it. The *medicinal dose* of Scheele's acid is from a minim to two minims,—of the London Pharmacopœial acid, from three to five minims gradually increased. The acid is said to float on the surface of water,—hence it has been advised to give it only in the form of draught: but when once mixed, this separation is not likely to happen. The variable proportions of strong acid present will, in some cases, explain the very different effects produced by equal quantities of different specimens. On the continent, the acid is met with of a strength rising as high as from ten to twenty-five per cent. The price at which the acid is sold to the public is about two shillings an ounce.

In giving an opinion on the quantity of this poison required to destroy life, it is material to know the variety of acid taken; and here it is much to be regretted, that in the British empire no uniform standard is adopted for so powerful a medicine. The following may be taken as the per-centage strength in anhydrous acid of the different varieties of this acid, British and foreign, in *aqueous* solution, on the authority of Dr. Christison and Dr. Pereira. Acid of Schrader, 1:—Dublin Pharmacopœia, 1·6 to 2·82 (Donovan:)—London Pharmacopœia, and the U. S. 2:—Göbel, 2·5: Edinburgh Pharmacopœia, 3·2:—Vauquelin and Giese, 3·3:—Scheele, 4:—Ittner, 10:—Robiquet, 50:—Among the *alcoholic* solutions of the acid,—Schrader, 1·5:—Bavarian Pharmacopœia, 4:—Duflos, 9:—Pfaff, 10:—Keller, 25 per cent.

[As above stated the acid made according to the formula adopted by the U. S. Pharmacopœia contains 2-100 of anhydrous acid, but much of that found in the shops is imported from Europe and is of variable strength.—G.]

Poisoning by prussic acid is commonly the result of suicide or accident. In 1837-8 there were twenty-seven cases of poisoning by this liquid, nearly all of which were the result of suicide. Within the last two years it has, however, acquired a fatal celebrity as a means of murder!

Effects of the vapour.—The vapour of anhydrous prussic acid, if respired, would prove almost instantaneously mortal. Even the vapour of the diluted acid accidentally respired, occasions very alarming symptoms. The following are the particulars of a case lately communicated to me by Mr. Tubbs: it may serve as a warning in the performance of experiments. A practitioner was showing to some friends the effects of Scheele's prussic acid on an animal, when, by accident, a quantity of the acid fell upon the dress of a lady who was standing before a fire. The poison was rapidly evaporated, and the lady was immediately seized with dizziness, stupor, inability to stand, and faintness. The pulse became feeble and irregular. Brandy was administered, cold affusion employed, and the patient was exposed to a free current of air. In ten minutes the pulse began to improve, and with the exception of tremor in the limbs, the unpleasant symptoms disappeared. I have known headach and giddiness produced by the vapour from the small quantities used in ordinary chemical experiments. Some caution is required even in temporarily smelling a bottle containing a strong specimen of acid. Chemical experiments show that this poison is always in the act of escaping from liquids which contain it; and the quantity evolved and diffused depends on temperature and the surface exposed to air.

Mr. Nunneley's experiments on animals prove that the action of the acid upon the lungs when air impregnated with the vapour is breathed, is not only rapid but certain in its effects, and it forms one of the easiest methods of exhibiting the poison—one which it would be very easy to employ, but after a few hours most difficult to detect, because the vapour, from its great diffusibility, is very soon dissipated. (Prov. Trans. N. S. iii. 84.) Baron Liebig asserts, what is wholly without proof, that this poison acts *only* through the vapour. He says, comparatively large (?) doses in aqueous solution may be taken into the digestive apparatus without producing any very perceptibly noxious effects, while the same quantity of acid inhaled as vapour, causes immediate death! Two drops of anhydrous acid in from four to six ounces of water produce no effect on a cat when swallowed. The same quantity of acid undiluted, placed on the tongue of a cat, causes no perceptible effect, provided the animal be prevented from breathing by stopping its mouth and nostrils, but the animal dies the instant it breathes, because the vapour then reaches the lungs. (Lectures in Lancet, Dec. 7, 1844, 306.) There can be no doubt that, like arsenic and all poisons, prussic acid acts much more speedily and powerfully when taken into the lungs as a vapour, than when introduced into the stomach as a liquid: but like other poisons it will also act through the mucous membrane of the alimentary canal. The experiment on the cat referred to by Liebig, is perfectly inexplicable; for admitting it to be possible to apply two drops of anhydrous prussic acid in the way described, and that a cat can live for a minute or longer with its mouth and nostrils perfectly closed, how is it to be explained that absorption should not go on, on the surface of the tongue as well as on that of the vagina or rectum?

I am not aware that there is any well-authenticated case of death having been caused by the vapour. The celebrated Scheele died suddenly while making his researches on this poison, and it is alleged that he was killed by respiring the vapour. The anhydrous acid was not known to Scheele; this was first prepared by Gay Lussac, in the early part of the present century.

In any case in which the poison is applied externally to the skin, the effects may become aggravated by the diffusion and respiration of the vapour.

Chronic poisoning by the vapour.—Dr. Chanet has lately called attention to the effects slowly produced by prussic acid vapour, upon those who are exposed to the fumes in a very diluted state. The process of galvanic gilding and silvering is now very common. Cyanide of potassium is used as a solvent for the metals, and as the solution is freely exposed, prussic acid is always passing off in vapour from its surface. This evolution of the vapour is aided by the warmth, and its noxious effects are aggravated by the closeness and want of ventilation in the rooms in which the process is carried on. The whole manufactory is perceptibly infected with the odour, and the workmen are thus compelled to breathe a poisonous atmosphere for many hours together. Dr. Chanet satisfied himself respecting the diffusion of the acid, by placing above the cyanide-bath, a watch-glass containing a solution of nitrate of silver. A white film of cyanide of silver was immediately produced on the surface. Some of the men are speedily obliged to abandon the work, from the feeling of illness produced. The symptoms in those who remain for a long time exposed to the vapour are:—dull headach, accompanied by shooting pains in the forehead, noises in the ears, vertigo, dizziness, and other effects indicative of cerebral congestion. Then follow difficult respiration, pain in the præcordium, sense of suffocation, constriction in the throat and palpitation, with alternate fits of wakefulness and somnolency. (Gazette des Höpitaux, 24 Juillet, 1847.) Dr. Chanet very properly asks the question,—whether by the introduction of the cyanide-process, as much mischief may not be done as by the mercurial process, which it superseded. For the local action of the Cyanide of Potassium, see post.

In trying some experiments on galvanic gilding, a few years since, the evolution of the prussic acid vapour was so manifest that nitrate of silver was whitened when exposed in the apartment at some distance, and the whole apparatus was therefore kept covered over.

From the researches of M. Gaultier de Claubry, it would appear that prussic acid is evolved in vapour to a dangerous extent in other manufactures. Some years ago a patent was taken out in France for the purpose of recovering the alcohol left in the residue of the preparation of the fulminate of mercury. Nitric acid and alcohol are here employed; and in addition to formic acid and hyponitrous ether, prussic acid is an invariable result. The proportion varies according to the degree of concentration of the liquids, but it is sometimes very considerable. In saturating the residuary liquid with chalk, prussic acid escapes in vapour, but the odour is in great part concealed by that of the ether. In distilling the liquid, prussic acid was abundantly obtained. On one occasion, the workman who stirred the chalk into the liquid, suddenly felt a severe pain in the head, his strength immediately failed him, and he fell down in a state of unconsciousness; a man who went to assist him was similarly affected. The experiment was performed in the presence of M. de Claubry, and he satisfied himself that these serious effects were due to prussic acid vapour, the odour of which was distinctly recognised. From only one drop of a portion of liquid distilled by himself, M. de Claubry suffered under the most alarming symptoms, from which he recovered after some hours. A fact of some importance is, that the alcohol thus recovered from the residue, which had been sold to the public for ordinary use, actually contains prussic acid! The public sale of this poisoned alcohol might account for many sudden deaths. A suggestion was made to the French Government to suppress it. (Ann. d'Hyg., 1839, ii. 350.)

Taste and odour.—The evidence derivable from the taste and odour of this poison is, in some instances, of importance. The *taste* is described by Dr. Christison as pungent; some state that it is hot, others that it is bitter. (Pereira.) When the common acid is taken mixed with organic liquids, the taste is not likely to be very perceptible unless the dose be exceedingly large. In *Belaney's* case the deceased swallowed the acid unmixed, and she cried out that she had swallowed a "hot liquid." We may probably trust to this statement as giving us the best idea of the taste of an unmixed and fatal dose of the poison. With a certain pungency there may be likewise a sense of bitterness, as in the bitter almond;—but it is obviously impossible to determine the exact kind of sensation produced on the tongue and fauces by a large dose of this poison. In an interesting case reported by Dr. Banks, a girl swallowed thirty drops: she called out for "bread," and on recovery in a few hours, she had no recollection of any thing that had transpired. A small but fatal dose of the diluted acid administered in a liquid like porter or in medicine, would probably have no perceptible taste.

With regard to the *odour*, Dr. Christison states that when diffused, it has a distant resemblance to that of bitter almonds: but it is accompanied with a peculiar impression of acridity on the nostrils and back of the throat. (Op. cit. 752.) Orfila also says that it is similar to that of bitter almonds:—this is, indeed, the common impression. There is, however, a difference between these odours; but the difference is not perceptible to the senses of all, and the only practical point requiring notice is, that the *diluted* odour of bitter almonds would probably be pronounced by many to indicate the presence of prussic acid, especially if there existed any suspicion of violent death. Even experienced medical men have to my knowledge been deceived on this point. There are some who are unable to perceive the odour of prussic acid, even when it exists in large proportion, whether mixed with water or other liquids; while others again are peculiarly susceptible of it. With some it does not affect the olfactory nerves at all; but produces merely a sense of constriction in the fauces. These facts appear to me to explain,—why on the post-mortem examination of a body, some persons may perceive the odour while others may not. When many have to form a judgment on this subject, it is much more common to find disagreement than unanimity. In the cases of the Parisian epileptics, three eminent physicians perceived no odour in the body twenty-four hours after death:—two others, equally eminent, profess to have perceived an odour of *bitter almonds* eight days after death! (Orfila, ii. 287.) In the case of Sarah Hart (*Reg.* v. *Tawell*, Bucks Lent Ass. 1845,) two medical witnesses perceived an odour on cutting into the integuments, a third could not: while not one of the four witnesses could detect any odour of the poison in the contents of the stomach or in the blood! In a case reported by Mr. Nunneley, two could perceive an odour about the nostrils of the deceased, in less than *six* hours after death, and two could not! (Prov. Med. Jour. July 23, 1845;) and in one lately communicated to me by Dr. Streeten, where death was recent and the dose large, five out of six medical gentlemen did not perceive any odour on approaching the body, either before or after it was opened.

On one point, however, there ought to be no difference of opinion. We should not rely upon the presence of a mere odour of "*bitter almonds*" as evidence of the presence of prussic acid; but either reject this altogether, or corroborate it by the application of tests to the suspected liquid in a separate state. In one instance (*Reg.* v. *Donellan*) the odour of bitter almonds was the only test; but there were peculiar moral circumstances in this case, which made up for the want of chemical evidence. All the soluble compounds of prussic acid, and the bruised kernels of many fruits, possess a similar odour; and it is important to observe, that there are some substances, containing no prussic acid, which may give out an odour like that of bitter almonds. This is observed in the compound called nitro-benzine. Dr. Skae perceived an odour of prussic acid on adding muriatic acid to meconate of lime, but he could obtain none of the poison by distillation. (North Jour. Med. May 1845.) Dr. Christison quotes two instances in which there was a strong smell of bitter almonds in the fæces, although no medicine containing hydrocyanic acid had been given to the deceased. (Op. cit. 775.) I have perceived it in the brain of a person who had died from ordinary disease, and whose body was examined soon after death. It is worthy of observation that the odour in these cases of artificial production, appears to be that of *bitter almonds*, not of prussic acid, and probably Dr. Skae refers to this odour in the above experiment. A bitter-almond odour may exist without the slightest trace of prussic acid being present, as where, for instance, the essential oil has been entirely deprived of prussic acid by chemical processes. This shows that the presence of the poison and this odour, are quite independent of each other. (For some remarks on this subject, see page 534, post.)

Cause of the loss of odour in the dead body.—The circumstances which may lead to the absence of odour *in a dead body*, in the contents of the stomach, or in any organic liquid, are—1, the smallness of the quantity of acid present; 2, volatilization by long exposure to air; 3, the smallness of the dose taken, and its entire removal by absorption and elimination when the individual has survived some time; 4, the degree of dilution of the poison, with water or other liquids; and, lastly, its concealment by other odorous bodies, such as vinous liquids; peppermint, or bitter almonds. (G. H. Reports, April, 1845.) Dr. Geoghegan detected the odour three days after death. Dr. Lonsdale found in his experiments on animals that the smell might be perceived for eight or nine days after death, although he could not detect the acid chemically for more than four days. (Ed. Med. and Surg. Jour. li. p. 52.) In the case of *Ramus* (Ann. d'Hyg. 1833, 365,) the odour was detected in the liquid distilled from the stomach *seven* days after death, and it yielded traces of prussic acid; but it was not perceived before distillation. This fact of the detection of the odour *after* but not *before* distillation, well known to all medical jurists, was absurdly made a point of great difficulty at the trial of *Tawell* for the murder of Sarah Hart, although the simple explanation is, that the poison is thereby separated more or less from other odorous substances which tend to conceal it.

In one instance in which three drachms had probably been taken, I found no odour in the stomach or its contents after twelve days. (G. H. Reports, April, 1845.) In a case examined by Mr. Hicks, ninety hours after death, where not more than nine-tenths of a grain had been swallowed and the individual died in twenty minutes, the contents of the stomach smelt strongly of prussic acid. It was also perceived on opening the cavity of the chest: but there was no odour of the poison about the mouth or in the room, although Mr. Hicks was present ten minutes before death! (Med. Gaz. xxxv. 893.) In a case examined by Mr. Pooley twenty-two hours after death, the smell was perceived in the stomach, but no where else. (Ib. p. 859.) In an interesting case communicated to the Lancet (Sept. 14, 1844,) by Mr. E. Crisp, of Walworth, in which a very large dose had probably been taken, he could detect no odour of prussic acid *in any part of the body*, although the inspection was made seventy hours after death: but a friend who was present thought he could perceive it. In a case in which a man swallowed nine-tenths of a grain and began to recover after four hours, the matters then for the first time thrown off the stomach had no smell of prussic acid. (Med. Gaz. xxxvi. 104.) Neither the dose nor the circumstances under which the body is exposed, will always suffice to explain these anomalies.

In the stomach of a dog poisoned by Mr. Hicks, three drachms of Scheele's acid having been introduced by the stomach-pump, the odour was perceptible to some persons, (but not to others) twenty-four hours after death: although the stomach had been laid quite open, freed of its contents, soaked in cold water, and placed for some time under a current of water. Mr. Hicks and I subsequently detected prussic acid in it with tests both with and without distillation. (Med. Gaz. xxxvi. 328.) This shows how completely organic matter becomes occasionally impregnated with the acid, but it is of course continually passing off in vapour—a fact proved by testing it in a way to be hereafter described.

Some objection was taken at the trial of *Tawell* to a very short abstract of a case by Mertzdorff, reported by me in a former work (Man. Med. Jur.,) because it tended to bear out what is beyond all question an established and undeniable truth, *i. e. the occasional disappearance of the odour of prussic acid in the dead body*. No odour was perceived in or about the body of a youth poisoned by three and a half drachms of an acid, five times the strength of that of the London Pharmacopœia, but it is said to have been perceived in the contents of the stomach. It is of itself sufficiently remarkable that with such a large dose of a very strong acid, the body and the cavities should have had no odour! (For a full account of the appearances in this remarkable case, see p. 536, post.) But in order to meet any future objections of so purely technical a character, I have here brought together a series of cases which, I think, will satisfy the reader of the truth of the *principle* for which alone Mertzdorff's case was briefly quoted, namely, the *uncertainty* of the evidence from the odour; and to these may now be added the case of *Sarah Hart*, and that communicated to me by Dr. Streeten,—all of them showing that to some persons very shortly after death, the odour may be perceptible, while to others it will be altogether absent. The object of attacking the terms of a mere reference to a case when the principle involved in the quotation was itself unassailable, was, of course, to divert the minds of the jury from the real facts. The question really before the Court was, Must the odour of prussic acid be always, under all circumstances and to all persons, perceptible in the dead body? In the face of good medical experience,—without reference to dose, the time of survivorship, vomiting, absorption, elimination, and admixture with other strongly smelling matters likely to be found in a dead stomach,—Sir Fitzroy Kelly, the counsel for the prisoner, contended that either the odour must be perceptible, or, as in respect to *Sarah Hart*, it was impossible to refer death to prussic acid! (For an account of this case, see page 543, post.) The numerous instances of the absence of odour which have been collected by toxicologists, most of which the reader will find recorded in this chapter, are, likely to prevent in future such a public misrepresentation of medical facts.

The odour of other substances mistaken for that of prussic acid.—A case which occurred in France in 1841, shows the great danger which may result from inferring the presence of this poison by the supposed odour. A. M. *Pralet*, æt. 64, was taken suddenly ill on the evening of the 13th January, 1841: he became insensible, and died six hours after the attack. The body was buried on the 16th, and exhumed on the 20th of the same month. Three medical men, assisted by a chemist (pharmacien,) undertook the investigation. As the result of their inquiries, they came to the conclusion that M. Pralet had been poisoned by hydrocyanic acid, and M. *Heritier* the nephew of the deceased, was charged with the murder of his uncle. It appeared in evidence that the deceased had dined as usual on the 13th of January, and that at eight o'clock in the evening he ate a small piece of bread and cheese, and drank three or four glasses of white wine. He was in the act of standing when he took the last glass, and he had scarcely swallowed it when he tottered, complained of illness, and became insensible. Various remedies were applied: he vomited a quantity of acid vinous liquid, recovered his consciousness, and said that he experienced no pain. Nevertheless his features were sunken, his face was pale, and his tongue and mouth were slightly drawn to the left side. In spite of remedial measures, there was a relapse. He became again unconscious and quite insensible to pain: the mouth was still more drawn on one side, and there was a tetanic stiffness of the left arm. The pulse, which up to this time had been full and regular, became weaker, and the patient expired at two o'clock in the morning, without having had any convulsions before death. The inspection of the body was made seven days after death. There was no putrid odour about it, but the face was discoloured. There was lividity of the skin in patches:—the subcutaneous veins were filled with black blood, and the nails were blue. On opening the abdomen, there was a peculiar odour, the nature of which could not be exactly specified: it was afterwards thought to be *more like* that of bitter almonds than any other substance! The stomach was collapsed, and the veins injected, especially towards the cardia: the heart and large vessels were empty, the lungs were soft, small, filled with dark blood, and having the same peculiar odour as that found in the abdomen. The surface of the brain was strongly injected with dark blood. On raising the hemispheres, a clot of the size of an egg was found:—this had also the peculiar odour. There was a large extravasation above the tentorium. The inspectors stated that a greater degree of cerebral congestion could scarcely have existed. An analysis of the contents of the stomach was made by applying tests to the distilled liquid. This liquid had a strong odour (not defined) which resembled that of prussic acid when sulphuric acid was added to it, and the mixture was diluted with twenty-three times its weight of water! The result of the application of the tests was, that in the opinion of the inspectors faint traces of prussic acid were present. (Annales d'Hyg. 1841, ii. 399, and 1843, i. 103, 474.)

The nephew was tried for the alleged murder, and narrowly escaped execution. An appeal (permitted in France in criminal cases) was made against the sentence, and the opinion of Orfila against death from poison, based on the facts of the report, became a subject of deliberation for three days. Orfila contended—1, that there was no proof, either from odour or chemical analysis, that prussic acid was present in the body; 2, that the symptoms and appearances were not like those met with in poisoning by prussic acid, but on the contrary, that they clearly indicated apoplexy from disease; 3, that death had been caused by apoplexy, and no crime had been committed. The result of the inquiry was, that the accused was discharged as perfectly innocent!

It is impossible not to assent to the correctness of Orfila's conclusions. The main ground of suspicion was, that the deceased had been attacked suddenly after taking a glass of wine. A remarkable case of death from an attack of apoplexy supervening on the taking of a small dose of sulphate of quinine has been elsewhere given (page 49, ante;) and it would have been just as reasonable to have attributed death to morphia in that case, as to prussic acid in this! The medical inspectors fell into the error of confounding a mere sequence with an effect. The symptoms were wholly unlike those caused by prussic acid; the partial paralysis, with the occurrence of a remission, showed the great probability of the attack being due to a cerebral lesion. The appearances were exactly such as would be found in a case of apoplexy, and unlike those caused by prussic acid. With respect to the *odour* observed in the body, the inspectors required some time to consider whether it was or was not like that of bitter almonds:—it could afford no evidence in so doubtful a case, unless the poison was clearly and unequivocally detected. The chemical evidence was, however, not merely defective, but in many respects completely erroneous;—the presence of any trace of prussic acid was not even rendered probable by the results. But for a proper medico-legal investigation of the facts, made by Orfila, M. Heritier, the nephew, would certainly have been guillotined! There are few cases in modern times which show more strikingly than this, on what a thread, the life of an innocent person, unjustly accused of the crime of poisoning, may depend. Courts of Law, both in France and England, wisely act upon the principle—*Cuique in suâ arte credendum;* but the above case shows that this confidence may be carried too far, and that special experience and special knowledge are absolutely required for the solution of these important questions.

Odour affected by saline solutions.—It become a question in *Belaney's* case (see post, page 541) how far the odour of prussic acid was likely to be concealed when the poison was mixed with a saline solution (sulphate of magnesia.) From experiment, I have not found that this made any other difference than mere dilution with an equal quantity of water. It is extremely easy to determine by chemical experiments the rapidity with which the vapour of prussic acid escapes. It is constantly evolved from all solids and fluids, but the evolution is slow in proportion to the degree of dilution.

SYMPTOMS.

The time at which the symptoms of poisoning *commence* in the human subject, is liable to great variation from circumstances not well understood. When a large dose has been taken, as from half an ounce to an ounce of the diluted acid, the symptoms may commence in the act of swallowing, or within a few seconds. It is rare that their appearance is delayed beyond *one or two minutes.* (For some remarkable instances in which their appearance was protracted, see p. 530, post.) When the patient has been seen at this period, he has been perfectly insensible, the eyes fixed and glistening, the pupils dilated and unaffected by light, the limbs flaccid, the skin cold and covered with a clammy perspiration; there is convulsive respiration at long intervals, and the patient appears dead in the intermediate time; the pulse is imperceptible; and involuntary evacuations are occasionally passed. The respiration is slow, deep, gasping, and sometimes heaving, or sobbing. The following case was communicated to me by my friend Mr. French:—it presents a fair example of the effects of this poison in a large and fatal dose. A medical man swallowed seven drachms of the common prussic acid. He survived about four or five minutes, but was quite insensible when discovered, *i. e.* about two minutes after he had taken the poison. He was found lying on the floor, senseless,—there was no convulsions of the limbs or trunk, but a faint flickering motion was observed about the muscles of the lips. The process of respiration appeared to cease entirely for some seconds:—it was then performed in convulsive fits, and the act of expiration was remarkably deep, and lasted for a very long time. The deceased swallowed the poison while ascending the stairs; his body was found on the landing. The bottle had rolled some distance from him, and the stopper was lying in another direction. Simon mentions a case in which an ounce was taken, and the symptoms were precisely similar. There was besides, coldness of the hands and feet; and no pulse could be felt. In such cases, *i. e.* where the dose is large, the breath commonly exhales a strong odour of the acid. Convulsions of the limbs and trunk, with spasmodic closure of the jaws, are usually met with among the symptoms; the finger-nails have been found of a livid colour, and the hands firmly clenched.

When a small dose (*i. e.* about thirty drops of a weak acid) has been taken, the individual has first experienced weight and pain in the head, with confusion of intellect, giddiness, nausea, a quick pulse, and loss of muscular power; these symptoms are sometimes slow in appearing. Vomiting has been occasionally observed, but it is more common to find foaming at the mouth, with suffusion or a bloated appearance of the face and prominence of the eyes. If death result, this is preceded by tetanic spasms, opisthotonos, and involuntary evacuations. Vomiting is sometimes the precursor of recovery. (See case, Med. Gaz. xxxvi. 103.) For an account of the symptoms produced by comparatively small doses, see cases by Mr. Hicks, (Med. Gaz. xxxv. 893,) by Mr. Pooley, (ib. p. 859,) and by Mr. Nunneley, (Prov. Med. and Surg. Jour. Aug. 13, 1845, p. 517.) The last case was remarkable in several particulars: the individual swallowed, it was supposed, forty minims of an acid, (at three and a quarter per cent.) and was able to give an account of his symptoms. He was conscious for some time after he had taken it, and he recollected experiencing the sensation of his jaws becoming gradually stiff and tight. It is not improbable, as Mr. Nunneley has suggested, that this poison may act more on the nerves of motion than of sensation, and that consciousness and sensibility may be retained by a person who has taken it, when from the powerlessness of the muscles, he is unable to indicate their existence. Prov. Trans. N. S. iii. 74.)

In an accident which occurred in Paris, seven epileptic patients in the Bicêtre were poisoned by an overdose of prussic acid administered in syrup. The quantity actually taken has not been satisfactorily determined. The symptoms as they are described by Orfila (Op. cit. ii. 286) and Devergie (Op. cit. ii. 281) were as follows:—Seven minutes after the poison had been swallowed, all the patients were found lying on their beds in a state of insensibility; they all had convulsions. The respiration was loud and hurried, the mouth covered with froth, the body in a state of perspiration, and the pulse frequent. To this state of general excitement, there gradually succeeded a mortal collapse. The act of respiration took place at greater intervals, and was of longer duration:—the pulse became weak and less frequent, there was cold perspiration, with coldness of the extremities, followed by death. In some, the skin of the head and face was strongly injected, in others it was pale; the pupils were moderately dilated; there was no vomiting; one of them made violent efforts to vomit a short time before he died. An attempt was made to put the feet in warm water, but most of them died before this treatment could be adopted. One who survived the longest, and who was thus treated, was shortly afterwards seized with very violent convulsions. He evidently felt the warmth of the water, for just before the convulsions came on his countenance expressed much suffering, and his breathing was more hurried. The face, conjunctivæ and head, became also strongly injected. A vein was opened, from which a small quantity of dark liquid blood escaped, but the patient speedily died. The first man died in from fifteen to twenty minutes, the last died in three quarters of an hour ofter having taken the poison. (For a further account of these cases, see page 535, post.)

It has been stated that those who died from this poison, uttered a *shriek* or *scream* as the last act of expiration. Such a symptom has never, so far as I can ascertain, been observed in the human subject. The cases in which persons have died from prussic acid, in the presence or in the hearing of others, are now very numerous. (See those of Mr. French, Mr. Hicks, Mr. Pooley, Mr. Godfrey, Mr. Nunneley, and Mr. Lowe, referred to in this chapter,) and in not one, was a shriek or scream observed to take place at any time! There was merely a gasping for breath, and a low moaning or sobbing noise, not more remarkable at the time at which insensibility supervened, than before. At the trial of Tawell, this was poetically described by the counsel in defence, as "the death scream;" as if it were uniform or even a common accompaniment of poisoning by prussic acid! This opinion appears to have gained ground from a misapplication of the effects occasionally produced on animals to the supposed action of the poison on man. Mr. Mills informs me, that in the course of his duties as deputy-coroner for Middlesex, he has had occasion to make inquiries into this subject; and the result is, that from the evidence of witnesses at inquests, there is not the slightest reason to believe that a shriek or cry before death, is a symptom attendant on poisoning by prussic acid in the human subject. All observers concur in the statement, that *convulsions* are almost invariably met with in animals destroyed by this poison. M. Bonjean remarked, that whether the acid was given in a concentrated state or diluted with water, the same train of symptoms was observed during the short period which preceded the death of the animal. All suffered from violent convulsive spasms of the limbs, with tetanic stiffness and abdominal respiration. When the action of the poison was once well marked, it went on until death. If a remission occurred, the animal could be restored to life by proper treatment. It sometimes uttered loud cries before death, probably from the pain experienced, and the body was rigid two hours after death, i. e. while it was yet warm. (Faits Chimiques rel. à l'Emp. par l'Acide Prussique, 1843, p. 34.) These results are in accordance with those obtained by Mr. Nunneley (Prov. Trans. N. S. iii.) and with those which I have had an opportunity of witnessing.

Effect of dilution.—It is necessary to consider how far the poisonous properties of the acid are affected by *dilution*. According to Baron Liebig, when the anhydrous acid is mixed with about 900 parts of water, it has no effect on a cat (ante, p. 518.) Some further experiments are, however, required, to determine whether dilution will thus disarm the poison of its virulence. In order that this should happen, water must have the power either of decomposing the poison, or of preventing its absorption. The former is contrary to all that is known of the properties of water, and the latter is opposed to facts. It has been well ascertained with respect to absorbable poisons, that by spreading them over a larger surface of mucous membrane, water rather tends to facilitate their absorption. The experiments made on animals by M. Bonjean and Mr. Nunneley, have clearly proved that prussic acid offers no exception to this rule. Dogs to which a diluted acid was given were quite as speedily and violently affected as those to which acid of Scheele's strength was administered. The same quantity of poison, therefore, may be expected to produce equally fatal effects, whether it be concentrated or moderately diluted. (Prov. Trans. N. S. iii. 82.) (As to the effect of *dose*, see post, p. 540, Period at which death takes place.)

Its effects contrasted with those of opium.—If we contrast the effects of this poison with those of opium, we shall find the following general differences. In opium, the coma comes on gradually, and is seldom seen until after the lapse of a quarter of an hour:—in poisoning by prussic acid, coma is almost instantaneously induced:—even in weak doses, insufficient to prove fatal, this symptom is rarely delayed beyond two minutes. In opium, the pupils are contracted, in poisoning by prussic acid, they are more commonly dilated. Convulsions are met with in both forms of poisoning, but perhaps more commonly in poisoning by prussic acid. With respect to the occurrence of this symptom, it is a fair question, whether medical jurists have not too readily adopted views, from the results of experiments made on animals—not from observations on man: since in very few instances, where the dose of poison has been *large*, has the patient been seen alive. When the dose has been small but still fatal, convulsions have been sometimes observed. A well-marked remission or intermission of the cerebral symptoms has been frequently noticed before death, in poisoning by opium (see ante, p. 469:) this has not been witnessed in poisoning by prussic acid,—the symptoms have been observed to progress in severity until death. In poisoning by prussic acid, the case, if fatal, generally terminates in less than an hour: in poisoning by opium, the average period of death is in from six to twelve hours. In poisoning by prussic acid, there is, in some instances, a smell of the poison about the mouth. Mr. Nunneley thinks, from experiments on animals, that if a person survive the first effects of the acid, the after-symptoms may be easily mistaken for those of opium. There is a deep quiet sleep, with difficulty of rousing the animal; and the pupils, in this stage, are not always dilated (Prov. Trans. N. S. iii. 76;) but, then, we must suppose that all other means of forming a diagnosis are wanting. The time at which the symptoms appeared after a liquid had been swallowed, their sudden invasion, the almost immediate loss of sensibility, and the odour of the breath, would, under ordinary circumstances, suffice to establish a diagnosis.

External Application.—Prussic acid is said to act through a wounded portion of skin. Sobernheim mentions the case of an apothecary at Vienna, who died in an hour from the entrance of the poison into a wound in the hand, produced by the breaking of a glass vessel in which it was contained. It is also said to act through the unbroken skin; but this certainly does not appear to be the case with the common diluted acid. The acid would doubtless produce all the effects of poisoning, if applied to an ulcerated or any highly absorbing surface. Mr. Nunneley ascertained in his experiments on animals, that the poison acted with the same rapidity and certainty on applying it to the mucous membrane of the conjunctiva, rectum, or vagina, as when swallowed. (Prov. Trans. N. S. iii. 84.) Dr. Christison states, that three drops of concentrated acid projected into the eye of a cat, acted on it in twenty seconds, and killed it in twenty more; and the same quantity dropped on a fresh wound in the loins acted in forty-five, and proved fatal in one hundred and five seconds. (Op. cit. 757.) See APPENDIX.

Accumulative properties.—A question has arisen, whether this poison possesses an accumulative power, *i. e.* whether, after having been taken in small doses and at short intervals, without apparent mischief, it may not suddenly give rise to all the effects of poisoning, either by a repetition of the same dose, or by a very slight increase in quantity. Dr. Lonsdale, who has examined the effects of the acid, does not admit that it possesses this property, on account of its great volatility and rapid diffusion. (Ed. Med. and Sur. Jour. li. 49.)

It appears reasonable to suppose that a poison which is so rapidly diffusible as prussic acid, should be speedily eliminated: it soon enters the blood, and, as it is well known, is expelled during life from the lungs. How long a period may be required thus to remove from the system a medicinal dose, it is not possible to determine; but if an interval of more than a day should occur, we may suppose that there would be no danger from the accumulation of the poison. Hence it is only a too frequent repetition of the acid, in innocent doses at short intervals, which is to be dreaded. Mr. Nunneley ascertained by his experiments, that where one dose, not sufficient to destroy life, had been given to an animal, a second and smaller dose, which by itself would not have killed, caused immediately violent symptoms and speedy death. There is one case reported, which renders the existence of an accumulative property, in the acid, to this limited extent, highly probable; and another has been communicated to me, which also bears out this view (p. 539.) The question is of considerable importance in respect to the medicinal use of the acid; for serious effects have repeatedly resulted from very slight alterations made in the dose.

The following case has a bearing on this question, and is of some interest, as it involved a respectable English physician, practising at Nice, in a charge of malapraxis. Dr. Gurney was called to a young lady who had been for some weeks in ill health, and was at the time suffering from severe spasmodic pain in the abdomen. He prescribed muriate of morphia and prussic acid: a sixth of a grain of the morphia, and a drop and a third of prussic acid (1·5 per cent.) at each dose. Fifteen doses were given throughout the day, making a total quantity of *two grains and a half* of morphia and twenty drops of diluted (= 3-10ths of a grain of anhydrous) prussic acid. The last dose was given fifteen hours before death. The patient at no time lost her consciousness or sensibility, nor did she suffer from any symptom indicative of narcotic poisoning. The whole of the prussic acid given at once would probably not have destroyed life, and although the total quantity of muriate of morphia might (if given at once) have proved fatal, it is impossible to refer death to it in the absence of any one symptom indicative of its effects. She answered questions, and was quite sensible, until within an hour and a half before her death, although thirteen hours had elapsed since the last dose was taken. (Med. Gaz. xxxix. 905.) The inspection revealed a sufficient cause of natural death, in disease of the liver and gall-bladder. Dr. Gurney was thrown into prison on a charge of some informality. The facts of the case was referred to Dr. Babington, Mr. Cooper, and myself: we agreed in certifying that there was not the least ground for imputing poisoning, but that death arose from natural causes. Dr. Gurney was subsequently liberated.

Period at which the symptoms commence. Power of Volition and Locomotion.—One of the most marked effects of prussic acid is to produce insensibility, and loss of muscular power, much more speedily than any other poison. In some instances, there may be loss of consciousness in a few seconds; in others, certain acts indicative of volition and locomotion may be performed, although requiring for their performance several minutes. This is one of the most important questions connected with death by prussic acid. In treating of this subject, Dr. Lonsdale says, that a drachm of Scheele's acid would affect an ordinary adult *within the minute;* and if the dose were three or four drachms, it would exert its influence within ten or fifteen seconds. When the acid is stronger and the quantity larger, we are pretty certain of its *immediate* action, and the consequent annihilation of the sensorial functions. (Ed. Med. and Surg. Jour. li. 50.) Mr. Nunneley found that in some instances the action of the poison was so expeditious as to prevent the least exhibition of voluntary motion: but in the majority *of dogs* about *twenty seconds* elapsed before any symptoms were manifested. (Prov. Trans. N. S. iii. p. 75.) Dr. Gerecke gave a tea-spoonful of concentrated prussic acid to a doe; symptoms were *instantaneously* produced, and in three seconds the animal was dead (Casper's Wochenschrift, 26, Sept. 1846, 615.) In the Leicester case (infra) Mr. Macauley found that a dog was killed in three seconds, and Dr. A. Thomson has observed that a dog has been killed in two seconds. Dr. Christison ascertained that a quantity of poison, equivalent to two scruples of medical acid, did not begin to act on a rabbit for *twenty seconds*, and certainly for so small an animal, two scruples are as large a dose as *five drachms* given to a grown-up girl. (Op. cit. 757.) These very different results appear to me to show clearly that experiments on animals cannot enable us to solve this question (ante, pp. 34 and 137.) We should rather trust to the few observations made on the human subject, as well as to analogy from other sources,—as, for example, to the fact of survivorship after the infliction of what are commonly regarded as instantaneously mortal wounds.

A case was communicated to me, by one of my pupils, where a man was found dead on the seat of a water-closet: he had died from prussic acid, and the bottle which had contained the poison, was found in his pocket, corked. Many similar facts are recorded, which show, that while as a general rule, insensibility may supervene from a large dose of this poison in a few seconds, the individual occasionally retains a power of performing certain acts indicative of consciousness, volition, and locomotion. In a case reported by Mr. Nunneley the man was enabled to speak rationally, and answer a question, after he had swallowed a fatal dose. (Prov. Med. Jour. July 23, 1845.) The importance of this question may be judged of by its bearing on the following case (*Rex* v. *Freeman*,) which was tried at the Leicester Spring Assizes, 1829. A full report of the case will be found in the Medical Gazette (vol. viii. p. 759.)

A young man, named *Freeman*, was charged with the murder of *Judith Buswell*, by administering to her prussic acid. The deceased was a maid-servant in the family of a druggist, to whom the prisoner acted as assistant. The deceased was one morning found dead in her bed: her death had been evidently caused by prussic acid, and it was presumed that she had taken *four and a half drachms* of the poison; the bottle out of which she must have drunk it, or had it administered to her, held an ounce, and it contained when found, three and a half drachms. Owing to the position of the body when discovered, and other circumstances connected with it, it was inferred that she could not have taken the poison herself. Her body was lying at length on the bed, the head being a little on one side. The bed-clothes were pulled up straight and smooth, and they came up to her breast;—her arms were under the clothes, and crossed over the chest. On turning the clothes aside, the phial which contained the poison was found lying on her right side. It was corked, and there was a piece of white paper round it,—the leather and string which appeared to have gone round the neck of the bottle, were found in the chamber-vessel. The medical question at the trial was,—Could this quantity of poison have been taken, and the deceased have retained volition and consciousness for a sufficiently long period to have performed these acts herself? Five medical witnesses were examined, and the opinions of four of these were strongly against the possibility of the acts having been performed by the deceased. One of the witnesses ascertained, that a dog to which the same quantity of acid was given, as was taken by the deceased, died in about *three seconds*. The medical opinion was founded on experiments of this kind; for there were no cases from the human subject, by which it could be supported. All of the acts to which the opinion referred, might be performed in from *five to eight seconds*; and there is nothing to warrant us in supposing, that under the above-named dose, all power would necessarily have ceased before this period of time had elapsed. On the contrary, there are now numerous facts which show that the symptoms may be often protracted for *several minutes.* Dr. Christison's experiment on the rabbit would lead to the inference that even five drachms would not begin to act upon a grown-up girl for twenty seconds (ante, 529;) and the results obtained by the witnesses from their experiments on dogs, were by no means uniform, even allowing that they were justified in applying them to the settlement of so important a question as this. The medical opinion was fortunately completely set aside by circumstances, and the prisoner was acquitted. A similar case occurred in Germany, and is quoted by Sobernheim. A young man swallowed four ounces of an acid (of four per cent.) equivalent to eight ounces of the pharmacopœial strength! He was found dead in bed,—the clothes drawn up to his breast, the right arm stretched out straight beneath the clothes, the left bent at the elbow-joint, and on each side of the bed, lay an empty two-ounce phial. There was no doubt of this having been an act of suicide. In this case more than three times as much acid was taken as in that of Buswell, but even here there was time for the performance of very similar acts! It is besides much more difficult to understand, how the poison should have been taken out of two phials, than out of one.

A few years since I had to examine a case of suicide by prussic acid, in which the facts were strongly confirmatory of the views here expressed. The deceased swallowed *three drachms* of prussic acid, and was found dead in bed, the clothes being smoothly drawn up to his shoulders, and there was no appearance of disorder about them, nor was there any sign of struggling before death. On a chair at the back of the bed, but close to it, was the phial which contained the prussic acid with the cork in it. (G. H. Reports, April, 1845.) There could not be the slightest doubt that the deceased had committed suicide, and that after swallowing the poison, he had retained sufficient sense and power to perform these acts. In a case reported by Mr. Crisp, the bottle with the stopper was found in the chamber-vessel, which had been pushed some distance under the right side of the bed, and here a very large dose had most probably been taken. (Lancet, September, 1844.) It has been supposed that under these circumstances of survivorship, the body should always be found convulsed, but this opinion is not borne out by facts. Here are cases of undoubted suicide, in which the body is found lying calm and tranquil without any mark of struggling or convulsions:—whether convulsions had taken place or not, is quite immaterial, since there was nothing to indicate that such symptoms had followed the ingestion of the acid. A very interesting case in reference to this question has been published by Mr. Leithead of Warkworth. A girl destroyed herself by prussic acid, and the evidence proved, so far as the facts could be proved, that she had swallowed *an ounce* of the acid, recorked the phial, thrust the bottle to a full arm's length between the feather-bed and the mattress,—got into bed, and then drawn the clothes over her body; there appeared to have been no convulsions. One medical man fancied he perceived the odour of prussic acid about the mouth, but another could not perceive it. (Lancet, June 7, 1845, 640.) This case appears to decide the question, i. e. that under a large dose without convulsions,—*insensibility or loss of consciousness may not come on until after the lapse of a sufficient time for the individual to perform acts which a few years ago, from experiments on animals, were deemed impossible!* Locomotion and muscular exertion are of course, compatable with small but fatal doses of this poison. In a case which occurred to Mr. Hicks, the girl sprang from her seat after swallowing the acid, threw her arms over her head, gasped for breath, and ran forwards about two yards before she fell. In one reported by

Mr. T. Taylor, the man ran twelve or fourteen paces before he fell, and remained insensible for a space of four hours,—a very long duration for the effects of this poison without causing death. Other cases recently observed, have shown that prussic acid does not give rise to insensibility and other alarming symptoms, so speedily as it was formerly supposed. Mr. Garson of Stromness has reported an instance in which a person for medical purposes, took at least a teaspoonful of prussic acid (the strength not mentioned;) the symptoms, however, did not come on for a *quarter of an hour*, when the patient was found insensible. He recovered, and stated that that period of time had probably elapsed between the taking of the dose and the commencement of the symptoms, and that he had employed himself in writing during the intermediate period! (Ed. Med. and Surg. Jour. lix. p. 72.) Perhaps one of the most extraordinary instances on record in this respect, is that related by Mr. Godfrey. A gentleman, æt. 44, swallowed, it was supposed, half an ounce of prussic acid, (strength not stated,) but certainly a quantity suficient to destroy life. After taking it from the bottle, he walked ten paces to the top of a flight of stairs, descended the stairs, seventeen in number, and went to a druggist's shop, at forty-five paces distance, where he had previously bought the poison, entered the shop, and said in his usual tone of voice, "I want some more of that prussic acid!" He then became insensible, and died in from five to ten minutes after taking the poison. This case is further of interest from the fact, that although it was an instance of *slow death*, there were *no convulsions*, there was no odour of prussic acid about the mouth, and the individual died in the presence of several medical men, without any shriek or any symptom approaching to it, being observed! (Prov. Med. Jour. Sept. 25, 1844.) Facts somewhat similar were witnessed by Mr. Nunneley, in a case reported by him in the same journal (July 23, 1845:) but in this instance the actual strength of the acid and dose swallowed, could not be ascertained. In two cases which have come to my knowledge within a recent period, in each of which a specimen of the acid was sent to me for the determination of its strength, this retention of volition and consciousness existed even after full doses of the poison had been swallowed. The first was a case which occurred at Worcester: the deceased must have swallowed at least *two grains* of anhydrous acid: he conversed, and manifested consciousness and volition for at least *two minutes*, and probably some time longer, after the poison had been taken. He died calmly; there were no convulsions, nor was any shriek heard. (For an account of this case, see Med. Gaz. xl. 171.) The second case occurred to Mr. Lowe. The quantity of anhydrous acid taken by the deceased, a young man æt. 23, was 2·54 grains. He had swallowed it in his bedroom:—he then descended thirty stairs, and walked about twenty paces before he became powerless. He was proceeding to open the front door of the house to go out, when he suddenly fell. The only symptoms observed by a person present were that "he threw his arms about, and made a noise in breathing, fetching it hard: he very soon became still." When seen by Mr. Lowe a quarter of an hour afterwards, there was no odour of prussic acid about the mouth or the body. (G. H. R. Oct. 1846, p. 490.) There is, I believe, no other instance recorded in which such a series of voluntary acts has been performed, and such a power of locomotion exerted, after so large a dose of the poison had been taken. It suggests, therefore, additional caution: it shows that full allowance must be made for the occurrence of some delay in the accession of insensibility and powerlessness, even when the dose of poison is large! The facts accumulated in reference to this question, are now so numerous and well authenticated, that it is never likely to become again a subject of doubt or dispute in a Court of Law.

Can a man after having taken prussic acid, live sufficiently long to attempt or *perpetrate suicide* in any other way? The following case occurred in London in April, 1839. A solicitor's clerk was found hanging quite dead at his chambers. He had evidently taken prussic acid; for a cup was lying near him which had contained this poison. The medical witness here very properly inferred, that the man did not swallow the acid until after he had adjusted the rope round his neck. It could hardly be admitted that a man should have power to hang himself, after having taken a large dose of this poison; but a person might be found drowned with prussic acid in his stomach, and without this fact being incompatible with suicide. It is however, a matter of doubt, determinable only by special circumstances, whether a man could or could not destroy himself by fire-arms after having swallowed the poison.

This question often presents itself in another form, namely, whether the act of poisoning was the result of *accident* or *suicide*,—this is of great importance when the life of the deceased happens to be insured. In general the circumstances are such as to explain at once the nature of the act; but a medical witness must remember that there is no instance in which suicide may be so secretly perpetrated by poison, as by prussic acid. There are besides many ways in which the means of death may be easily concealed; and as it is so little the custom to cause an inspection to be made of the bodies of those who have died suddenly, unless a suspicion already exists of death having been caused by poison, such cases might easily escape detection. (See Borough Inquests, 1845, by Dr. Birt Davies.) In the event of litigation ensuing, in respect to a policy of insurance, it may be too late to discover any traces of poison in the body. It is proper therefore to remark, that the effects of prussic acid may be easily confounded with the symptoms of all diseases which are liable to destroy life suddenly; such as epilepsy, apoplexy, and diseases of the heart (p. 61, ante.)

The case of *Mrs. Maclean*, who was found dead in her house at Cape Coast Castle, in October, 1838, is interesting to the medical jurist, in relation to the question of suicide or accident. Her attendant, in going to the room of the deceased, found some difficulty in opening the door, in consequence, as it appeared, of the body of the deceased having fallen against it. The deceased was lying on the floor, quite senseless, with an empty bottle in her hand, uncorked, and labelled "hydrocyanic acid, medium dose five minims." There was a feeble pulsation of the heart, which soon ceased. It appeared that the deceased was in the habit of taking prussic acid as a medicine; and the medical witness supposed, that she might have taken an overdose, and have thus been killed accidentally; he was so fully convinced that the medicine was the cause of death, that he did not open the body. By this omission, the case was left in mystery; for had the body been inspected, and the larger portion of the contents of the bottle been found in the stomach, there would have been no doubt of its having been an act of suicide; since a well-informed person like the deceased, was not likely to have swallowed by accident a large dose of a poison, with the deadly properties of which she must have been perfectly acquainted. If the acid had been taken medicinally, and an overdose swallowed by accident, it is singular that the bottle should have been found in her hand; since we cannot suppose that any educated person would take a medicinal dose of prussic acid conjecturally, by swallowing it from the bottle itself! In consequence of this omission to inspect the body, it is now difficult to say whether this was an act of suicide or the result of accident.

POST-MORTEM APPEARANCES.

The body often exhales the odour of prussic acid when seen soon after death; but if it has remained exposed for some time before it is seen, and especially if it has been exposed to the open air or in a shower of rain, the odour may not be perceptible. In a case in which a person poisoned himself with two ounces of the acid, and his body was examined twenty-eight hours after death, the vapour of prussic acid which escaped on opening the stomach was so powerful, that the inspectors were seized with dizziness, and obliged to quit the room hastily. This may serve as a caution in conducting an examination. In cases of suicide or accident, the vessel out of which the poison has been taken will commonly be found near; but there is nothing to preclude the possibility of a person throwing it from him in the last act of life, or even concealing it, if the symptoms should be protracted. Putrefaction is said to be accelerated in these cases; but from what I have been enabled to collect, there seems to be no ground for this opinion, any more than in the case of poisoning by opium. (See case by Mr. Nunneley, Prov. Med. Jour. July 30, 1845.) Orfila has shown that in most instances of *sudden death* from whatever cause, putrefaction is, *cæteris paribus*, accelerated; and the fact that in one or two instances of death from prussic acid, the bodies have speedily putrefied, has improperly led to this condition being set down as one of the characters of poisoning by this acid.

The post-mortem appearances are very slight. *Externally*, the body is commonly livid, or the skin is tinged of a violet colour; the nails are blue, the fingers clenched, and the toes contracted; the jaws firmly closed, with foam about the mouth, the face bloated and swollen, and the eyes have been observed to be glassy, very prominent and glistening, but this condition of the eyes exists in other kinds of death. *Internally*, the venous system is gorged with dark-coloured blood: the *stomach* and alimentary canal are in their natural state; but in some instances they have been found congested or inflamed. The mucous membrane of the stomach of a dog which died in a few minutes from a dose of three drachms of Scheele's acid, was intensely reddened throughout, presenting the appearance met with in cases of arsenical poisoning. In a large number of experiments upon dogs, Mr. Nunneley found that there was generally a congested condition of the mucous membrane of the stomach: if empty at the time the poison was taken, the organ was found much contracted, and of a brick-red colour. The same appearance of congestion was observed on the mucous membrane of the vagina, the rectum, and conjunctiva, when the acid was applied to those parts. (Prov. Trans. N. S. iii. p. 79.) The same redness was observed in the cases of the Parisian epileptics (see post, p. 536;) and Dr. Geoghegan of Dublin has recently communicated to me the particulars of a case in which this redness of the stomach was well marked. In April 1847, a healthy man, æt. 30, swallowed a large dose of prussic acid. He was soon afterwards found dead in his bed. The body was inspected five hours afterwards: rigidity had commenced, but there was some warmth. The face was pale, the eyes half-closed, not presenting any remarkable brilliancy or prominence, nor was there much dilatation of the pupils. The mouth was closed, and no froth issued from it. The abdomen was the only cavity examined. The muscles were red, and gave out, on section, a good deal of fluid blood, which had a strong odour of prussic acid; the odour of the poison was also perceptible in the peritoneal cavity. About eight ounces of a thick farinaceous mass were found in the stomach: the odour of prussic acid was very perceptible in this organ, but it was mixed with that of rancid food. The mucous membrane had every where, except at the splenic end and posterior wall, a vivid inflammatory redness, of a well-marked character, and it was, to a considerable extent, lined with a layer of viscid mucus. The parietes were not thickened, but the submucous coat presented ramified vascularity; the peritoneal coat was also decidedly red. The posterior wall, at the splenic end, was of a chocolate colour, with scattered petechiæ: the great venous trunks stood out in relief as dark blue lines. The mucous membrane, even when washed three times in water, gave out a strong odour of prussic acid.

The *odour* of the poison, if not observed in the body, is generally perceptible in the stomach for several days after death, unless the quantity of poison be small, and it be mixed up with other strongly smelling substances. (On this subject, see ante, p. 521.) If death has been rapid, the dose large, and the inspection recent, as in the case just related, all the cavities as well as the blood have the odour. Besides these appearances, the brain and lungs have been found congested, although not invariably. The blood is, in some instances, quite liquid, in others, thick and semi-coagulated. (Heller's Archiv. i. ii. 1845, p. 143.) In most cases this liquid has been found of a very dark colour,—in a few, red (Heller's case, supra,) and in other cases again of a violet or pinkish hue. Heller found, by a chemical and microscopical examination, that in one instance the blood contained no fibrin (supra.) In two instances, reported by Mertzdorff, the contents of the gall-bladder had a blue tint, but this appearance may have been owing to accidental causes, as, in the generality of cases, there has not been observed any abnormal change in the bile. The larynx, trachea, and œsophagus, are said to have been found reddened; but it is not impossible, that this redness may have depended on other causes. Death commonly takes place with such rapidity, as scarcely to allow of the production of any well-marked morbid changes in the body. In a case reported by Dr. Geoghegan, where a man swallowed an ounce of prussic acid, and was found dead, the only morbid appearance of note discovered, was a patch of dark red extravasation, under the mucous membrane of the stomach near the pylorus. The stomach in this case, exhaled the odour of hydrocyanic acid, although it had been exposed for *three days*, but the poison was easily detected, in its contents, by the usual processes. In a case reported by Mr. Pooley, a dark colour of the blood appears to have been the only striking appearance (Med. Gaz. xxxv. 859: in this instance the lungs were not congested, in a case by Mr. Hicks, they were much congested (Med. Gaz. xxxvi. 460,) while in Mr. Nunneley's case they were only partially congested. (Prov. Med. Jour. July 30, 1845.) In the case reported by Mr. Crisp (Lancet, Sept. 14, 1844,) the abdominal and thoracic viscera were healthy, with the exception that they had a purple colour from the blood: he could perceive no odour of the poison. From this general summary of the appearances, it will be perceived, that there is but little to be derived from an inspection of the body, at all characteristic of the mode of death: and probably in many instances no suspicion of the cause would be excited, except for the occasional presence of the well-known odour.

Special cases.—In a case which occurred to Mr. Ilott, in which probably death had been occasioned by a large dose, there was slight congestion of the cerebral vessels, but the organ was otherwise healthy, and there was no effusion. The lungs were congested, and the large veins of the chest filled with blood; the lining membrane of the œsophagus was intensely reddened; the stomach in various points, but more especially near the œsophagus, was inflamed or congested; the mucous coat was softer than usual, but corrugated. There was a distinct smell of prussic acid in the contents. (Med. Times, June 6, 1846, 197.)

In July, 1847, Dr. Streeten communicated to me the particulars of a case in which an adult died in five minutes from a dose equivalent to about *two grains* of anhydrous acid. The following appearances were met with:—Countenance (particularly the lips) livid; neck, shoulders, and all the posterior part of the trunk, purple. On dividing the integuments, dark blood flowed freely; the blood of the body universally dark and quite fluid; the lungs loaded with dark blood, which had to a considerable extent gravitated to their posterior portion. The right auricle and right ventricle of the heart, and vena cava, were full of dark fluid blood; the left ventricle was firmly contracted and quite empty. Five out of six gentlemen did not perceive any odour of prussic acid upon approaching the body, either before or after it was opened. All the abdominal viscera were healthy; the urinary bladder was half full of urine, which exhaled no unusual odour; the brain natural, but full of fluid blood. The stomach contained about one ounce of raspberry-coloured looking fluid, and had a strong smell of almonds. Traces of prussic acid were discovered in it by distillation. The stomach itself, particularly at its cardiac extremity, had a very vascular appearance, and in some of the patches, oozing of blood had evidently taken place, while in others the mucous membrane had a brownish appearance.

A very complete account of the post-mortem appearances observed in the bodies of the seven patients accidentally poisoned in a Parisian hospital by an overdose of prussic acid, has been published by Orfila. (Ann. d'Hyg., 1829, i. 507.) For a more full description of the symptoms, see ante, page 525. The inspection was made by MM. Adelon, Marc, and Margolin.

X., æt. 15, was seized with convulsions in about eight or ten minutes after he had taken an overdose of hydrocyanic acid syrup: he then fell into a state of collapse, and died in half an hour. It was observed in this, as well as in the six other cases, that the symptoms were marked by two distinct periods,—one spasmodic or convulsive, indicative of irritation—the other of collapse, or relaxation; exactly like those observed in dogs poisoned by the acid. An inspection of the body was made twenty-four hours after death. No external mark of violence, excepting a slight redness on the left instep, produced during the convulsive fit; lividity of the back; the head, face, and lips violet, and slightly swollen; a frothy sanguineous liquid issuing from the mouth and nose; the mouth so firmly closed, owing to the general rigidity of the body, that it could not be opened; the eyes were closed, and on separating the lids, the pupils appeared somewhat dilated. On making an incision into the muscles they were found of their usual colour. The interior of the mouth, fauces, and œsophagus presented nothing remarkable. The sub-peritoneal cellular tissue of the stomach and small intestines was deeply injected, without, however, being ecchymosed or inflamed. At ten inches from the ileo-cœcal valve, the injection was so strongly marked as to give to the intestines a black colour. This appearance was not met with in the large intestines; they were healthy, and contained a small quantity of gas. The alimentary canal being properly secured by ligatures, was removed for the purpose of collecting the contents. Those of the stomach were placed in a vessel, and this organ was then examined;—*no particular odour* was remarked; here and there were patches of redness, especially on the rugæ; towards the pyloric extremity the mucous membrane had a granular appearance, owing to the enlargement of the mucous glands, but there was no sign of ecchymosis or erosion. Red patches, due to capillary injection, with enlargement of the mucous glands, were observed in the small intestines. At the part of the small intestines corresponding to the black patch above referred to, a small quantity of blood was found extravasated beneath the mucous and muscular coats. The large intestines were quite healthy, and contained fæcal matter of the ordinary colour and consistence. The liver, spleen, and kidneys were natural, excepting that they contained a larger quantity than usual of dark-coloured blood. The membrane covering the kidneys was easily detached. The bladder was healthy, and contained urine. The heart appeared natural, there was no change of colour, or any alteration in its substance: it was quite empty, containing neither fluid nor coagulated blood. The larger arteries were likewise empty, but the veins, on the contrary, were filled with liquid blood of a dark colour. The lungs were of a reddish colour anteriorly, and slightly congested posteriorly. The mucous membrane of the larynx, trachea, and larger bronchi, was of a deep red colour, and the deeper ramifications of the bronchial tubes contained a frothy sanguineous liquid. On raising the scalp, a moderate quantity of blood escaped. The sinuses of the dura mater were full of a dark fluid blood. The substance of the brain presented no mark of inflammation or ecchymosis,—it was softer, and its vessels were more congested, than usual. The spinal marrow, examined throughout its whole length, was quite healthy.

From observations made at the inspection of the whole of the seven bodies, the appearances, in different degrees of intensity, were of the following kind:—

A decidedly inflammatory condition of the mucous membrane of the stomach and small intestines, with enlargement of the mucous glands, and a slight injection of the subperitoneal cellular tissue. The spleen softened, in some cases almost pulpy; the veins of the liver filled with black liquid blood; the kidneys of a deep violet colour, softened, congested, and easily denuded of their investing membrane; the heart firm in substance, but perfectly empty, as well as the larger arteries,—the large veins, on the contrary, full of black liquid blood,—the blood itself every where fluid, and in no part presenting the least trace of coagulation. The mucous membrane of the larynx, trachea, and bronchi, was of a deep red colour, not removeable by washing, and the bronchial tubes filled with a frothy bloody liquid. The membranes of the brain were injected; the sinuses of the dura mater congested with black liquid blood; the substance of the brain softer than natural, but otherwise healthy, as well as the spinal marrow. No part of the body gave out the odour of bitter almonds, *i. e.* prussic acid; nor were there any signs of putrefaction; and in all the subjects, there was a remarkable degree of cadaverous rigidity. The mucous membrane of the bladder was white and healthy, as well as that of the pharynx and œsophagus. Such is a summary of the principal appearances.

An interesting case has been reported by Mertzdorff, in which the dose of poison taken was unusually large, and the post-mortem appearances were accurately noted. A young man poisoned himself with three drachms and a half (Orfila wrongly describes the dose as three *grammes and a half*, ii. 285) of Ittner's prussic acid (containing ten per cent. of anhydrous acid) = *twenty grains* of anhydrous acid. He was found dead in bed. The body was examined thirty hours afterwards. At this time it was already somewhat decomposed, the scrotum being of a livid colour, and in places deprived of its cuticle; several livid-red spots were observed on the face, about the chest, neck, and shoulders; there were also a few vesicles on the left thigh. The body did not emit any odour of bitter almonds (prussic acid.) The pupils were neither dilated nor contracted; the teeth were not closed together; the tongue was immediately behind them. The abdomen was not distended. The limbs were somewhat flexible, the nails blue, and the fingers bent. The stomach and greater part of the intestines could be readily torn, the former contained a viscid mass having the odour of prussic acid; its internal surface was of a bright red colour and streaked with blood, especially in the neighbourhood of the cardiac and pyloric orifices. The mucous membrane of the intestines was also reddened in many places. The liver, spleen, and kidneys were of their natural colour, and were loaded with fluid blood of a blueish-black colour (schwarz-blau.) The bile was of a dark blue colour. The bladder and pancreas were both healthy. The muscles generally were of a darker colour than natural. The lungs were healthy in structure, but had the same blueish-black colour as the blood. The right and left cavities of the heart were filled with fluid blood containing a few coagula. There was no serum in the pericardium or in either pleural cavity. The tongue was not red, but the larynx, trachea, and œsophagus, as far down as the stomach, were of a blueish-black colour, and the trachea contained a large quantity of blood. The brain was healthy, but its vessels and sinuses were loaded with blood, so that it streamed forth when the scalp was cut into and removed. (Horn's Archiv. für Med. Erfahrung, 1823, ii. p. 55.)

After this description of the appearances met with in death from a very large dose of the poison, it may be proper to state those found in the body of an adult female killed by the smallest dose of prussic acid yet known to have destroyed life—*nine-tenths of a grain.* The inspection was made ninety hours after death. The teeth were clenched, and foam was still adhering round the mouth; the face was of a dusky-red hue, and the whole of the depending part of the body of a dark purple or violet colour: it had very much the appearance of the body of a person who had died from asphyxia. The dura mater and sinuses were much congested, and the whole of the substance of the brain was dotted with blood, which was fluid and very black; the ventricles were empty, and the plexus choroides pale and bloodless; but no odour of prussic acid was perceptible. On opening the chest, the odour was more plainly perceived than in any other part of the body; the lungs were much congested, but otherwise healthy; the right ventricle of the heart was distended with fluid black blood. The stomach contained four ounces of fluid smelling strongly of prussic acid: its lining membrane was healthy, with the exception of a small patch of redness near the cardiac orifice: but as the deceased had suffered from gastric symptoms, this may not have been due to the action of the poison. The liver, gall-gladder, and kidneys, were healthy, except that the latter were congested, and had a dark pinkish hue. (Med. Gaz. xxxvi. 460.)

In a case communicated to me by Mr. Newham, in which a man had died from the effects of three drachms of prussic acid, the following appearances were found:—The membranes of the brain were perfectly natural in every respect: the substance of the organ was also firm and natural. The quantity of serous fluid contained in the ventricles was less than usual; but it was strongly impregnated with the odour of prussic acid. The plexus choroides were pale and bloodless. The lungs were healthy: at the upper part, the organs were of a light red colour; at the lower part they were full of a dark-coloured, muddy-looking blood. The heart was natural, but contained very little blood: this was of a dark and muddy hue, and strongly impregnated with the odour of the poison. The liver was larger than natural, and there were several spots of medullary deposit about it. The gall-bladder was nearly empty, and the bile was dark-coloured. The pancreas was healthy, and the spleen, externally, was of a very bright violet-purple colour. The urinary bladder was natural: it contained no urine. The stomach contained more than half a pint of a viscid liquid, having a strong odour of prussic acid. A portion of the mucous membrane, at the greater curvature, was highly injected and inflamed, being of a deep red hue. The intestines were healthy, but generally empty; the mucous membrane of the duodenum was slightly inflamed in patches; and on being laid open, there was a strong odour of the poison. The lower portion of the great intestines was quite empty, the deceased having involuntarily passed the fæces and urine, while under the influence of the poison, and probably in the act of dying. (G. H. Rep., April, 1845.)

QUANTITY REQUIRED TO DESTROY LIFE.

This is a very important question; and it is made somewhat perplexing by the fact, that beyond a certain dose, the weak and the strong acid appear to act with equal rapidity. (Christison, 658.) It has been already stated, (ante, p. 137,) that six drops have been found to destroy the life of an animal as rapidly as one ounce of the same acid; the animals being alike in strength and vigour. (See also Pereira, Mat. Med. i. 439.) If any inference could be drawn from these experiments applicable to the human subject, it is clear that the view often adopted, of the rapidity of death being in proportion to the largeness of the dose, is erroneous. This statement may be true to a limited extent; but it appears to me that a most improper application of it has been made from a few experiments. Admitting that two drachms of Scheele's acid will certainly kill an adult within a given period, it by no means follows that four times that quantity will kill a similarly constituted person within one-fourth of that period. Yet this has been made the basis of evidence by medical witnesses, as if it were a positively established fact. I have sought through works on toxicology, and our periodical journals, in vain, for cases by which such a view could be supported. It is directly opposed to what we observe in the action of other poisons; for it is in general impossible to say within what time a case will prove fatal, from the actual quantity of poison taken. In one instance related by Dr. Geoghegan, a quantity of the acid, equal to twenty-seven drops of the English pharmacopœial strength, (at two per cent.) was taken by a gentleman without any effect, the dose having been gradually raised to this point; but no bad consequences whatever had resulted from the acid previously taken. He now raised the dose to thirty-six drops, and in two minutes he was seized with the usual symptoms, and nearly lost his life. (Dublin Med. Jour. viii. 308.) The quantity of anhydrous acid, (0·66 grain) swallowed in this dose, was less than three-quarters of a grain, *i. e.*, about equal to eighteen drops of Scheele at four per cent. Cases hitherto observed show that there is a very narrow line between the quantity of the poison which may be taken with impunity, and that which is required to produce death. In determining the quantity necessary to prove fatal, we must, it appears to me, for the purposes of legal medicine, avoid the results obtained by experiments on animals, and look to those facts only which have been ascertained from observation on the human subject. In general the quantity taken is extremely large; but the *smallest* dose known to have caused death was in the female whose case is reported by Mr. Hicks. (Med. Gaz. xxxv. 896.) The patient, a healthy woman, died in twenty minutes from a dose equivalent to *nine-tenths* of a grain of anhydrous prussic acid. This was equivalent to *forty-nine drops* of the London Pharmacopœial acid, and taking Scheele's acid at four per cent., (Pereira,) to about *twenty-five drops* of Scheele. In an interesting case reported by Mr. T. Taylor, (Med. Gaz. xxxvi. 104,) a stout healthy man swallowed this dose, *i. e.*, nine-tenths of a grain, by mistake, and remained insensible for *four hours*, when he vomited and began to recover. The vomited matters had *no odour* of the poison, showing that if not concealed by other odours, the whole of the acid must have been here absorbed. He had a very narrow escape of his life. Dr. Banks has published a case in which a female recovered after swallowing thirty drops of prussic acid, (Ed. Med. and Surg. Jour. xlviii. p. 44,) but the interest of this case is lost, owing to the strength of the acid not having been determined. It is advisable to put no trust in any statements bearing upon an important question of this kind, where *a direct analysis of part of the poison swallowed* has not been performed. Serious mistakes may be made in deciding on the strength of a fatal dose, by the *average* strength of the variety of acid that has been taken;—since what is called Scheele's acid may vary from two to five per cent. In the reports of cases it would be advisable that the dose taken, should always be expressed in the quantity of *anhydrous* and not of the diluted acid. In the case lately referred to me for analysis by Dr. Streeten, the acid had been sold to the deceased as of Scheele's strength. After taking a large dose, the patient manifested powers of volition and locomotion for five minutes. This would have been extraordinary had the acid been really Scheele's, as the patient would have swallowed about *five grains* of anhydrous acid. On analysis I found it to contain rather less than *two* per cent.

By trusting to this mode of calculation, all English medico-legal writers have been misled with respect to the dose taken by the seven Parisian epileptics (p. 535.) Dr. Christison, Dr. Lonsdale, and Dr. Geoghegan had stated the dose which here proved fatal, at about two-thirds of a grain. A similar dose was assigned to these cases in the first edition of my "Manual of Medical Jurisprudence." The mode in which the mistake arose is explained by Dr. Lonsdale. (E. M. and S. J. loc. cit.) Some reflections have been made upon English writers in relation to this error; but it has been overlooked or concealed,—that Orfila himself, who has been made the standard of accuracy, gives two different versions of the dose;—Devergie gives another account, and Guibourt a fourth! (See Med. Gaz. xxxv. 896; also Pharmaceutical Journal, May, 1845, 515,) and the only inference which a medical jurist can draw from all this confusion, is, that it is now utterly impossible to assign the real dose taken in these cases; because no direct analysis appears to have been made of any portion of the hydrocyanic acid syrup actually *swallowed;* and it appears to me that we cannot trust to any evidence short of this in an important question of this kind.

Recoveries from large doses.—The *largest* dose from which an adult has recovered, was probably in the case reported by Mr. Nunneley. (Prov. Med. Jour. Aug. 13, 1845, p. 517.) The person swallowed, it was supposed, *forty minims* of an acid at three and a quarter per cent. Taking the minim as equal to the grain, although it may be a little more or less according to circumstances (see p. 540,) this is equivalent to about *one grain and one-third* of anhydrous acid. The man was for a short time conscious, got into bed after taking the poison, and spoke. He felt his jaw become stiff and then remained insensible, until roused by the cold affusion. The fact of recovery having taken place here, must not lead us to suppose that such a large dose could be commonly taken with impunity. If we refer to the chapters on arsenic and corrosive sublimate, we shall find that persons have recovered from doses of these poisons, much larger than those which have proved fatal in other cases. The same circumstance is observed in respect to all other poisons. Judging by the effects produced in Dr. Geoghegan's case from 0·66 grain of anhydrous acid,—from the fact that death took place in Mr. Hicks's case from nine-tenths of a grain; and that, in another instance, a strong adult had a narrow escape of his life from the same dose, we shall not be wrong in assuming that a quantity of Scheele's acid (at five per cent.) *above twenty drops*, (i. e. *one grain of anhydrous acid*,) or an equivalent portion of any other acid, would commonly suffice to destroy the life of an adult. This I believe to be the nearest approach we can make to the *smallest fatal dose*. Even less than this—seven-tenths of a grain, might under favourable circumstances prove fatal. We have no certain evidence from recorded facts, that two grains of arsenic have yet destroyed the life of an adult; but the probability is, that this quantity would be a fatal dose. Neither seven-tenths of a grain of Prussic acid, nor two grains of arsenic might suffice to kill *every* person; but this is not the kind of information which the law requires. A witness is only required to say what dose will, under ordinary circumstances, commonly suffice to destroy the life of an adult: age, idiosyncrasy, habit, state of health, and fulness or emptiness of the stomach, make the same difference here as in the case of other poisons. (See Opium, ante, p. 481.)

In estimating doses, it may be proper to state the results of some experiments on the weight of given measures of this poison. Sixty drops of a P. L. acid, or one drachm, measured in a drop measure, commonly used in a laboratory, weighed 64 grains; 60 minims in a measure used in the dispensary department of Guy's Hospital, weighed 61 grains; and the same quantity in a measure procured from a large retail druggist's, weighed 62·5 grains. One drachm in a common two-ounce measure, weighed 68 grains, and one ounce in the same measure balanced, weighed 444 grains, which gives an average weight of 55·5 grains for each drachm. In another instance a quantity which destroyed life was equivalent to 105 drops, and weighed 98 grains. To ensure accuracy these experiments wereperformed by double weighing; and the results show how little certainty there is, with respect to the exact quantity of the anhydrous acid in minim and drop doses. The stronger the acid, the less the weight of a given bulk, since the specific gravity diminishes in proportion as the water becomes saturated with the pure anhydrous acid. An ounce of distilled water should weigh at 62° 437·5 grains.—A drachm should weigh 54·7 grains, and a minim or drop, 0·91, or 91-100ths of a grain.

PERIOD AT WHICH DEATH TAKES PLACE.

Some remarks have already been made on this subject, and it has been particularly stated that beyond a certain point, we are not entitled to infer, that the rapidity of death bears any proportion to the quantity of poison taken (p. 538.) Experiments on animals might be adduced to prove either the negative or affirmative of this proposition,—a fact which clearly shows, that they cannot be safely admitted in this particular, as a basis for medical evidence. They prove that the fatal operation of the acid is not always rapid in proportion to the quantity of poison taken. Dr. Reid found that dogs of the same size and strength died in the same period of time, whether the dose was *an ounce* or only *six drops* of Scheele's acid (ante, page 137,) the quantity given being no less than seventy times greater in the one case than in the other! Mr. Nunneley states as the result of his observations on animals that there is no well-marked difference as to the period at which this poison destroys life between doses differing much from each other, provided each be capable of destroying life with moderate rapidity. (Prov. Trans. N. S. iii. 83.) It has been clearly observed in the human subject, that where individuals have taken the same dose, death has occurred at very different periods of time: age, idiosyncrasy, the state of health, and the presence or absence of food in the stomach, are conditions which of course exert an appreciable influence on the operation of this poison. In the cases of the seven Parisian epileptics (ante, p. 526) a *similar* dose was given to all, but death took place at very *different* periods,—the first person died in about fifteen or twenty minutes, and the last, only after three-quarters of an hour! In one instance in which seven drachms of the acid were taken, death took place within five minutes:—in another, in which an ounce was taken, the individual survived about ten minutes. (Sobernheim.) When the dose is two drachms and upwards, we may probably take the average period for death at from *two* to *ten minutes*. In Mr. Hicks's case, forty-nine drops of P. L. acid destroyed life in twenty minutes. It is only where the dose is just in a fatal proportion, that we find the individual to survive from half an hour to an hour. In this respect, death by prussic acid is like death by lightning:—the person in general either dies speedily, or recovers altogether. According to Dr. Lonsdale, death has occurred in the human subject as early as the *second*, and as late as the *forty-fifth* minute. But although death does not commonly ensue until after the lapse of a few minutes, insensibility, and consequently a want of power to perform acts of volition and locomotion, may sometimes come on in a few seconds (see ante, p. 528.) The time at which this loss of power is supposed to take place, has frequently become an important medico-legal question; and on the answer to it, the hypothesis of suicide or murder in a particular case, may rest.

TREATMENT.

Experience justifies us in employing stimulants, such as diluted ammonia to the nostrils, and frictions of the compound camphor liniment to the chest. Chlorine has been strongly recommended as an antidote to be injected in the state of solution into the stomach; but admitting that it were at hand to be administered in a case of poisoning which seldom lasts above a few minutes, it is a remedy of very doubtful character. Lately, Messrs. Smith of Edinburgh have proposed the mixed oxides of iron as an antidote,—these forming on contact with prussic acid, insoluble Prussian blue. Ittner and Chancel long since recommended a mixture of sulphate of iron and potash. (Lonsdale, loc. cit.) The jaws are, however, commonly so firmly closed, that it is difficult to make the individual swallow any thing, and the poison operates with fatal rapidity. The iron-antidote is only likely to be useful when it is employed early, and the poison has been taken in a very diluted state. Mr. Nunneley found that it had no beneficial effects on dogs. (Prov. Trans. N. S. iii. 87.) It has been proposed to apply electricity in the course of the spinal marrow; but the best remedy, and that which is always applicable, is *cold affusion*. This has been found the most efficacious mode of treatment in experiments on animals, and in several cases in the human subject. One of these is reported by Dr. Banks of Louth. A girl took by mistake in medicine, *thirty drops* of prussic acid. Immediately afterwards she sprang up convulsively from her seat, and became senseless. Her teeth were firmly set, and her eyes staring and fixed. Stimulants failed to rouse her:—the limbs became flaccid;—the pupils dilated, and she was wholly insensible; the respiration was slow, and the pulse scarcely perceptible. A stream of cold water from a pitcher was allowed to fall from some height on the region of the spine. In a minute she began to move, and became convulsed; her symptoms abated, and in a few hours she was quite collected. She recovered in a few days, but there is hardly a doubt that she would have died, had she not been thus treated. (Ed. Med. and Sur. Jour., xlviii. 44.) The following interesting case of recovery, even where cold affusion was applied late, is reported by Mr. T. Taylor (Med. Gaz. xxxvi. 104:)—H. G., æt. 59, a healthy agricultural labourer, rather stout made, swallowed by mistake a dose of Pharmacopœial acid equal to nine-tenths of a grain, in eighteen drachms of distilled water. He had no sooner taken it than he was seized with a violent constriction of the diaphragm, with a sense of suffocation. He walked to the outer door of the house, about twelve or fourteen paces, when he fell insensible, and, in his fall, broke a large pan which was full of water, the contents of which saturated his clothes, and this, no doubt, had a beneficial influence upon him. The accident occurred about half past seven o'clock in the morning, and it was not until 20 minutes after eleven, or nearly four hours after taking the acid, that he showed symptoms of returning animation, when, by the application of cold water and ammonia, he was soon restored. On coming to himself, he vomited freely, but no odour of the acid was perceptible in what he threw off his stomach. The next day he was well. Mr. Harthill has also reported a case in which cold affusion led to recovery after probably a large dose. (Prov. Med. Jour., March 5, 1845, p. 153.) In Mr. Garson's case (Ed. Med. and Sur. Jour., lix. 72, and p. 531, ante,) cold affusion and the use of ammonia appear to have been attended with the best effects. Bleeding from the jugular vein is strongly recommended by Dr. Lonsdale. It is likely to be beneficial in protracted cases when there is any sign of cerebral congestion; but, in general, the pulse is scarcely perceptible, and this mode of treatment is therefore inapplicable. There is always great loss of power under the operation of this poison: hence, the imprudent or hasty abstraction of blood may actually retard recovery, or even accelerate death (p. 526.) Mr. Nunneley's experiments on dogs show that this mode of treatment is not attended with any benefit. (Prov. Trans. N. S. iii. p. 72.)

[From the experiments of Simon, Orfila and others, it would appear that chlorine is an antidote when promptly employed, and the same may be said of ammonia, though it is not as certain. But as no time is to be lost in these cases, it is better to resort at once to the cold affusion. Venesection should never be used to combat the primary symptoms, though it may be sometimes required in the treatment of secondary affections.—G.]

Cases.—In the case of *Reg.* v. *Belaney* (Cent. Crim. Court, Aug. 1844,) some important questions arose respecting the proper mode of treating cases of poisoning by prussic acid. The prisoner was a surgeon, and he was charged with the murder of his wife, who died in his presence from the effects of a large dose of prussic acid. The medical facts in the case were very simple. There could be no doubt that the poison had been taken, and that it was the cause of death. The nature of the symptoms, their rapid and fatal course, and the detection of the poison in large quantity in the stomach, rendered these conclusions absolutely certain. Again, there could not be the smallest doubt, that the poison had been administered either intentionally or unintentionally by the prisoner, *i. e.* that it was through his act, either criminal or innocent, that the poison was placed within reach of the deceased; and under circumstances which would render it not improbable that she would swallow it by mistake. It was placed in a common drinking-glass in the bedroom,—the prisoner being at the time in an adjoining room. The prisoner accounted for this circumstance, by saying that he was in the habit of using prussic acid, medicinally,—that he broke the bottle in trying to remove the stopper; and, in order to save the contents, collected the acid in a tumbler or glass, such as is used for drinking water! His attention was called off, and he went into an adjoining room, without, as it would appear, making any remark, or cautioning his wife respecting the poison placed in the tumbler, and within her reach.

The presumption of criminality, under such circustances, had no direct relation to medical evidence: it was a question to be decided by the jury from the facts proved. The medical evidence had, however, two important bearings: 1, the plan of treatment which should be adopted in such an emergency, by a medical man; 2, the exact period at which insensibility and loss of consciousness supervene in cases of poisoning by prussic acid.

The prisoner, on finding that his wife had swallowed the poison, called for assistance, but did not at the time state the real cause of the symptoms; although it came out in evidence that he must have known that the deceased had swallowed prussic acid. He caused her feet and hands to be put into hot water, and talked of bleeding her, but said it was of no use, as circulation had ceased ("she had no pulse.") He told the first witness who came to her, that "she would not come to,—it was a disease of the heart, and that her mother had died just like it nine months ago:" but it was subsequently proved that the prisoner had himself registered the cause of death in the mother, as bilious fever; Dr. A. T. Thomson, who gave evidence at the trial, was questioned upon the usual remedies in such cases, which he stated to be, cold affusion, ammonia and stimulants, and very properly expressed an opinion, that what had been done by the prisoner, could be of no benefit whatever.

The second question related to the power of the deceased to talk after she had called out or screamed, as deposed to by her husband. The prisoner said that he heard a cry (probably of alarm,) and on going into the bedroom, his wife exclaimed, "I have taken some of that hot drink, give me some water." Dr. Thomson was inclined to believe, that upon the utterance of a cry of this description, volition and sensibility would be lost, and it would afterwards be impossible for a person to talk. This observation was derived from experiments on animals. He admitted, however, that the cry might really have proceeded from an unpleasant sensation experienced by the deceased on swallowing the acid, and not have been connected with the loss of sensibility or consciousness. In charging the jury, the judge properly stated, that an inference on this point, not being derived from the observation of the effects of the poison on man, should be received with caution. The jury acquitted the prisoner. The verdict did not proceed from any defect in the medical evidence; the cause of death was clear, and it was for the jury to determine the value of the moral and circumstantial evidence against the prisoner as the administrator. Of these circumstances, which were exceedingly strong, it is here unnecessary to speak: but the jury, in the opinion of most persons, took a very lenient view of them.

Some remarks have been elsewhere made concerning "a shriek" alleged to take place in poisoning by this acid, (ante, p. 526.) The counsel for Belaney, in addressing the jury, said, "that the effect of prussic acid was to cause a shriek, and with that shriek, volition ceased altogether." Dr. Thomson informed me subsequently to the trial, that he had only in some instances observed, in poisoning animals by prussic acid, that death was preceded by a peculiar vocal sound resembling "a cry of pain," a statement which, with the cases already reported, will tend to remove an erroneous impression, that the utterance of a scream or shriek commonly attends this form of poisoning. Numerous cases are now reported, which show that it cannot be regarded as a symptom in the human subject.

Among the cases which have drawn attention to the subject of poisoning by prussic acid, there are few which have excited greater interest than that of *Sarah Hart*. (*Reg.* v. *Tawell*, Bucks Lent Assizes, 1845.) A very good report of this case by Dr. Skae has been published in the Northern Journal of Medicine for May, 1845. From the medical evidence it appeared that the deceased, a healthy woman, was found dying. The practitioner who was called in, bled her, but without effect. On a post-mortem examination, eighteen hours after death, two surgeons out of three who were present, perceived in cutting into the integuments, what they thought was an odour of prussic acid; but no odour was perceived about the mouth, or in the blood which had been drawn from the body. The viscera were generally healthy. The lungs were slightly congested, and there were some old pleuritic adhesions, but there was no lesion of any organ to account for death. The stomach and bowels presented no morbid change. The contents of the former amounted to twelve ounces of liquid, *having no odour* of prussic acid, but merely a strong acid smell of beer. (Lancet, April 5, 1845, 379.) They consisted of partially digested food, intermixed with the pulp of apple. Prussic acid was obtained from the contents of the stomach by distillation: it was identified by the application of the usual tests, and after separation as cyanide of silver, by its odour. The quantity thus obtained amounted to *one grain* of anhydrous acid! The administration of the poison to the deceased, was clearly brought home to the prisoner, partly by a series of moral circumstances of a most convincing kind, and partly by his own admissions, attributing, however, the death to suicide. Suicide was entirely out of the question.

Space will not permit me to give a lengthened analysis of this case, but it may be proper to point out a few of the most remarkable medical assumptions made by Sir F. Kelly, the learned counsel who conducted the defence. They were,—1. That there must be *direct* evidence of death from poison! 2. That the evidence of medical witnesses who had never before met with a case of poisoning by prussic acid, should not be received! 3. That the *quantity* of poison *found in the stomach*, should be sufficient to cause death! "If there was not enough prussic acid in the stomach to account for death, there was an end of the case." The fact is notorious that the poison found in the stomach is always the surplus quantity, and not that which has occasioned death. (See ante, p. 117.) 4. That although a *grain* of prussic acid (anhydrous) was there found, yet the witnesses had not proved *upon their own knowledge and experience*, that that was sufficient to cause death! (See cases, p. 538, ante.) 5. That because persons had recovered from a larger quantity than was found in the deceased's stomach, it was not to be inferred that she had died from the effects of a grain! 6. That a grain of anhydrous prussic acid might have been spontaneously produced in the stomach of the deceased, partly from the pips of apples, a portion of cake, the saliva swallowed during mastication, and partly from the decomposition of animal matter.

The judge summed up the case in a manner which showed that he was perfectly acquainted with all the fallacies of such a defence. The statement of the prisoner's counsel that it was a rule of law, that there should be direct proof of death having been caused by poison, and of the presence in the stomach of a sufficient quantity of poison to produce death was not true,—neither was it necessary to prove what quantity of prussic acid would destroy life by the testimony of a person who had actually seen a human life destroyed by it. With regard to the *smell*, the only conclusion from the evidence was, that smell was a proof of the presence of the poison, but that the absence of smell was no proof of its absence. According to the witnesses, a grain or even less than a grain of prussic acid taken into the stomach, was sufficient to cause death. With respect to the allegation that prussic acid might be obtained from apple-pips, Mr. Cooper, the chemical witness, found apple but no pips in the stomach, and it was only by the distillation of the pips that the acid was formed, whereas the odour of the poison was perceived in the body by two witnesses. After a short deliberation, the jury returned a verdict of guilty, and the prisoner before execution confessed that he had perpetrated the murder in the manner in which it had been proved against him.

It is unnecessary to repeat what has been elsewhere stated with respect to the value of evidence from the odour of the poison. (See ante, p. 520.) The argument of the learned counsel was founded upon an obvious misapprehension of medical facts. It is to be regretted, however, that he took upon himself to pronounce as false those reports of cases published in medico-legal works, which went to show that owing to various circumstances the odour of prussic acid might not be always perceptible in the stomach of a person poisoned by it. Thus Dr. Christison had stated that it was not perceived in the stomachs of the Parisian epileptics. Sir F. Kelly told the Court, on the authority of Orfila, that this was an error, and that the odour was there perceived. The facts have been elsewhere stated, pp. 520, 536. MM. Adelon, Marc, and Marjolin, made the inspection, and they plainly declare that they could detect no odour of bitter almonds, (*i. e.* of prussic acid) although the inspection was made only *twenty-four hours* after death, and the dose taken was assumed by Sir F. Kelly to have been much greater than represented by Dr.

Christison and myself. There was no odour in the stomach, its contents, or in any part of the body. (Ann. d'Hyg. 1829, i. 507.) Instead of quoting this plain statement made by those who inspected the body, the learned counsel put forward the opinions of Gay Lussac and Orfila, who were *not present* at the inspection, that there was an odour of bitter almonds in the contents, although they did not examine them until *eight* days after their removal from the body. This apparently conflicting opinion, first published by Orfila fourteen years after the event (Toxicologie, ii. 287,) was adduced by Sir F. Kelly as the only true representation of the facts at the trial. The unfairness of such a mode of dealing with medical reports must be apparent; but it becomes even more glaring from the following extract of a paper, published by Orfila four years *before* this trial, in which, in a disputed case of death from prussic acid, he makes use of the fact here misstated and perverted, to show that the odour of prussic acid may be really absent in the dead body! In reference to the case of *Pralet* (ante, p. 523,) where too great a reliance upon odour nearly led to the execution of an innocent person, Orfila says,—"What confidence can be placed in the odour of bitter almonds, said to exist in the body of the deceased, when we find that MM. Marjolin, Marc, and Adelon declare that no part of the bodies of the Bicêtre epileptics exhaled the odour of bitter almonds?" (Ann. d'Hyg. 1841, ii. 409.) From this it must be obvious that Orfila considers, on the authority of those who were best cápable of judging, that there was no odour perceptible in these cases, and, therefore, while the truth of Dr. Christison's statement was wrongly impugned, an opinion was imputed to Orfila which he did not entertain! (See APPENDIX.) Compare also the case of *Rumus* (Ann. d'Hyg. 1833, p. 365.)

But the whole of the defence was involved in a mass of contradictions. The learned counsel, by attempting to account for the existence of *a grain* of anhydrous prussic acid in the stomach through its spontaneous generation from apple-pips, admitted its *presence* in the contents. In making this admission he could not escape from another, *i. e.*, that this acid was there *before* distillation; because at the time of distillation no apple-pips were found in the contents! As he assumed that no odour was perceptible on inspection, he must therefore have admitted that this large quantity of prussic acid might be present without being indicated by smell; and yet the basis of his argument in the defence was—because there was no odour, there was no prussic acid present! Such contradictions must always follow when an attempt is made to argue against incontrovertible facts.

An opinion has gone abroad, that the poison in this case was not administered by the mouth, but in a more secret way. This opinion is based on the absence of odour in the stomach, and on the reported confession of the criminal. It is founded, however, on a mistaken view of the medical facts. One grain of anhydrous prussic acid would not have been found in the stomach, had not *more* than this quantity been actually swallowed: but this dose was of itself sufficient to destroy life.

CHAPTER XXXVIII.

PRUSSIC ACID, CONTINUED. CHEMICAL ANALYSIS—IMPROVED PROCESSES—TESTS FOR THE ACID IN THE SIMPLE STATE—AS A LIQUID AND IN VAPOUR. SILVER, IRON, AND SULPHUR TESTS—OBJECTIONS TO THE TESTS—THEIR COMPARATIVE DELICACY—DETECTION OF PRUSSIC ACID IN ORGANIC LIQUIDS—PROCESS WITHOUT DISTILLATION—OBJECTIONS—DETECTION BY DISTILLATION. ABSORPTION OF PRUSSIC ACID—ITS DETECTION IN THE BLOOD AND TISSUES—CAUSES OF NON-DETECTION—COMPARATIVE VALUE OF CHEMICAL EVIDENCE—ALLEGED SPONTANEOUS PRODUCTION OF THE ACID—QUANTITATIVE ANALYSIS.

CHEMICAL ANALYSIS.

PRUSSIC ACID is limpid like water; it possesses a faint acid reaction, and its vapour has a peculiar odour, (ante, p. 520,) which when the acid is concentrated, although not at first perceptible, is sufficient to produce giddiness, insensibility, and other alarming symptoms. (See ante, p. 518.) Until within the last few years it was supposed that the odour might be present in cases in which it would be impossible to detect the poison by chemical processes, and instances of this are given by Orfila, (Ann. d'Hyg. i. 489,) by Dr. Lonsdale, (Ed. Med. and Surg. Jour. li. 52,) and by Dr. Christison, in the last edition of his Treatise. (Op. cit. 1845, pp. 760, 774.) Improved methods of applying tests for the detection of the diffusible *vapour* of the poison, instead of attempting to separate it, as a liquid, by distillation, have, however, established the contrary position, namely, that the poison may now be detected in cases in which its odour cannot be perceived. The fallacy which was therefore attempted to be imposed on a Court of law as an ascertained fact, even so recently as 1845, is now completely exploded, and is not likely to be revived in any future case. (See the case of *Reg.* v. *Tawell*, ante, p. 543; also Med. Gaz. xxxvi. 328, 588, and 631.)

The tests which are best adapted for the detection of this poison, either in liquid or vapour, are equally applicable whether the acid be concentrated or diluted, and, so far as the detection of the *vapour* is concerned, whether it be pure or mixed up with organic matter.

TESTS IN THE SIMPLE STATE.

The tests are three in number:—the *Silver*, the *Iron*, and the *Sulphur* tests.

1. *Nitrate of Silver.*—This yields, with prussic acid, a dense white precipitate, speedily subsiding in heavy clots to the bottom of the vessel, and leaving the liquid almost clear. The precipitate is identified as cyanide of silver by the following properties:—*a.* It is insoluble in cold nitric acid, but when drained of water, and a sufficient quantity of strong acid is added, it is easily dissolved on boiling. It has been asserted that it is soluble in *cold* nitric acid, but this only applies to those cases in which the quantity of precipitate is so small as to be scarcely ponderable, and the proportion of nitric acid is very large. Under ordinary circumstances, cold nitric acid dissolves scarcely any perceptible portion, and thus easily distinguishes the cyanide from the oxalate, carbonate, and some other salts of silver. *b.* The cyanide is slighty soluble in a large excess of nitrate of silver: hence, when the acid is in small quan-

tity, a very small portion of the test should be added. *c.* It is soluble in a large excess of solution of potash, if there be some free hydrocyanic acid present:—it is also soluble in ammonia, but this is by no means a distinctive character, and therefore is unimportant. *d.* It evolves prussic acid when digested in muriatic acid. *e.* The precipitate, when *well dried* and heated in a small reduction-tube, yields cyanogen gas, which may be burnt at the mouth with a rose-red flame and blue halo. This is a well-marked character, and at once identifies the acid, which yielded the precipitate, as prussic acid. By this property the cyanide is eminently distinguished from the chloride, oxalate, and all the other salts of silver. In a small tube, three-fourths of a grain of the precipitate will thus furnish good evidence. *f.* As an additional means of identifying this precipitate as the *cyanide* of silver, even when the quantity is so minute that the experiment of combustion cannot be satisfactorily performed, Lassaigne advises that it should be converted to cyanide of potassium, by heating it in a very small reduction-tube, with a small piece of potassium—the cyanide being dropped on the metal. When cold, cut off the end of the tube, digest the residue in a few drops of distilled water, and add to the liquid, 1, a solution of green sulphate of iron and muriatic acid, and 2, a solution of sulphate of copper. The production of a blue colour in the first case, and of a red in the second, will indicate that cyanogen must have been present in the precipitate. (Ann. d'Hyg. ii. 121.) Orfila states that this process will detect with certainty half a milligramme (1-130th of a grain) of cyanide of silver, and this is equal to the 1-750th part of a grain of anhydrous prussic acid. Dr. Austin, of Dublin, has more recently recommended that oxide of iron and carbonate of potash should be mixed with the precipitate and treated in a similar way. (Dub. Hosp. Gaz. April 1, 1846..) Fine iron-filings may be substituted for the oxide, but in all cases potassium, if it can be obtained, is preferable. These processes are open to a serious fallacy. Any kind of organic matter, provided it contain nitrogen, will when heated with potassium or with iron and alkali, yield a soluble ferrocyanide. *g.* If, instead of burning the gas produced by heating the precipitate, it be received on the recently precipitated mixed oxides of iron (see IRON TEST,) containing a little potash, Prussian blue will be formed, and made evident by soaking the paper in diluted muriatic acid. *h.* Lastly, the most conclusive means of identification, when the quantity is small, is to place the suspected precipitate in a watch-glass, moisten it with concentrated muriatic acid, and absorb the vapour by hydrosulphuret of ammonia. (See SULPHUR TEST.) The vapour may be also received on potash, and the iron-test applied. (See IRON TEST.)

In the employment of the silver test for the detection of the *vapour* of the poison, we place a few drops of the silver solution in a watch-glass, and invert it over another watch-glass containing the suspected poisonous liquid. Cyanide of silver, indicated by the formation of an opaque white film in the solution, is immediately produced, if the acid be only in a moderate state of concentration. One drop of the pharmacopœial acid (containing 1-50th of a grain) produces speedily a visible effect. When the prussic acid is much diluted a few minutes are required, and the opaque film begins to show itself at the edges of the silver solution. In this case the action may be accelerated by the heat of the hand.

2. *The Iron Test.*—The object of the application of this test is the production of *Prussian Blue.* We add to a small quantity of the suspected poisonous liquid a few drops of potash and of a solution of green sulphate of iron. A dirty green or brownish coloured precipitate falls: on shaking this for a few minutes, and then adding diluted muriatic or sulphuric acid, the liquid becomes blue; the Prussian blue, of its well-known colour, unaffected by diluted acids, subsides. If the prussic acid be in very small quantity, the

liquid is at first yellow, from the salt of iron formed; it then becomes green, but the precipitate ultimately subsides so as to appear of a blue colour in the mass. The same result is obtained, by adding the solution of the iron-salt to the potash-solution of the cyanide of silver; and thus, in this way, the two tests may be applied to only *one* portion of the poison. The great value of the iron-test is, that it will equally act in cases where the results of the silver-test would be obscure, as where the prussic acid happens to be mixed with any other acid or salt precipitable by the nitrate of silver, *e. g.* with muriatic acid or an alkaline chloride. Mr. Ilott, of Bromley, has shown, that when a very small quantity of prussic acid is mixed with a large quantity of caustic ammonia, the iron test acts somewhat ambiguously, especially when muriatic acid is used to redissolve the oxide of iron. I have found, however, that the ambiguity only exists when these different substances are in certain relative proportions, and it is difficult to conceive a case in which this effect would become a practical interference with the inference from the test. Besides, any difficulty would be at once removed by the application of the silver and the sulphur tests.

The iron test may be employed for the detection of the *vapour* of prussic acid, by the same method as that described in speaking of the silver test. For this purpose we place a few drops of caustic potash in a small white saucer, and invert it over the suspected liquid. After a few minutes a drop of solution of green sulphate of iron may be added, and then a drop of diluted muriatic acid,—Prussian blue appears. The recently precipitated mixed oxides of iron with potash, may be placed in the upper vessel with the same results. The silver and the iron tests may be in this way easily conjoined in testing the same quantity of poison. If the precipitated cyanide of silver, obtained by the addition of nitrate of silver to the suspected liquid, be moistened with strong muriatic acid, and the vapour collected in a watch-glass or saucer, on the plan just described, Prussian blue will be procured, and thus strongly corroborate the action of the silver test.

The Sulphur Test.—Mr. Porrett informs me that, in the year 1814, he discovered a method of producing sulphocyanic acid by the mutual reaction of prussic acid and an alkaline sulphuret. Baron Liebig has recently proposed this as a process for detecting prussic acid as a liquid. (Oesterreichische Med. Wochenschrift, 27 März, 1847, 396.) If a small quantity of hydrosulphuret of ammonia (containing a little excess of sulphur) be added to a few drops of the solution of prussic acid, and the mixture be gently warmed, it becomes colourless, and, on evaporation, leaves sulphocyanate of ammonia—the sulphocyanic acid being indicated by the intense blood-red colour produced on adding to the residue a solution of a persalt of iron; this colour immediately disappears on adding one or two drops of a solution of corrosive sublimate. (See ante, p. 503.) This test is very delicate, but it requires a little care in its application: thus, if the boiling and evaporation be not carried far enough, the persalt of iron will be precipitated black by the undecomposed hydrosulphuret of ammonia; and, if the heat be carried too far, the sulphocyanate of ammonia may itself undergo decomposition, and be lost. It will be perceived, too, that it requires a longer time for its application than either the silver or iron test.

The great utility of the *sulphur test*, however, is in its application to the detection of the minutest portion of prussic acid when in the state of *vapour*. In this respect it surpasses any other process yet discovered. In order to apply it, we place the diluted prussic acid in a watch-glass, and invert over it another watch-glass, holding in its centre one drop of the hydrosulphuret of ammonia. No change apparently takes place in the hydrosulphuret; but if the watch-glass be removed after the lapse of from half a minute to ten minutes, according to the quantity and strength of prussic acid present, sulphocyanate of ammonia will be obtained on gently heating the drop of hydrosulphuret and evaporating it to dryness. With an acid of from three to five per cent. the action is completed in ten seconds. The addition of one drop of persulphate of iron to the dried residue, brings out the blood-red colour instantly, which is intense in proportion to the quantity of sulphocyanate present. Such is the simple method of employing the test. When the prussic acid is excessively diluted, the warmth of the hand may serve to expedite the evolution of the vapour.

In detecting the vapour, the *sulphur test* acts, *cæteris paribus*, more rapidly and more delicately than the *silver* test; but the two may be usefully employed together in corroboration of each other. If a suspected liquid, placed in a watch-glass, produce a film on a drop of nitrate of silver, the reaction will be very speedy with the hydrosulphuret. The silver test acts *visibly*, and therefore serves to guide us; the sulphur test acts invisibly; for there is no apparent change unless the glass be left so long that the ammonia is spontaneously evaporated, and the sulphur oxidated or deposited. If the suspected liquid produce, after half an hour, no effect by its vapour on a drop of nitrate of silver, it is very probable that no prussic acid is present: nevertheless, we should proceed to apply the hydrosulphuret of ammonia, as the poison may still be detected by it when nitrate of silver entirely fails to indicate its presence. The sulphur-test is applicable even to the detection of decomposed prussic acid. It was tried with a specimen which had been kept three years:—it was quite black, and in a thick and syrupy condition. This liquid produced by its volatility an opaque film in a few seconds, and the prussic acid still remaining in it, was detected in an equally short period by the hydrosulphuret. If the hydrosulphuret of ammonia have absorbed no prussic acid, it gives before evaporation a black precipitate with persulphate of iron; if it be evaporated until the yellow colour has disappeared, should no sulphocyanate be present, the salt of iron produces merely a yellow tint from the separation of sulphur. Other alkaline sulphurets may be substituted for the hydrosulphuret of ammonia, but they must be always in the state of polysulphuret: and the chemical reaction with prussic acid is never perfect, until after heat has been applied to the mixed liquids, or to the sulphuret which has absorbed the vapour.

Other tests and processes have been suggested for the detection of prussic acid. Thus *Protonitrate of Mercury* gives with prussic acid a deep blackish-grey precipitate. It thereby clearly distinguishes it from all acids, including the muriatic, which gives with the test a white precipitate (calomel.) This test acquires a dark-grey colour with prussic acid, even in a very diluted state, *e. g.* in laurel water and bitter almond water. Calomel may be substituted for the protonitrate: the effect is the same. If prussic acid be present in minute proportion, calomel acquires a dark-grey colour. In 1839, Mr. York, a former pupil, suggested that an aqueous *solution of iodine* might serve as a test. The brown colour of this solution is immediately removed, and the liquid rendered colourless, by a very minute portion of prussic acid. The colour of bromine in water is similarly discharged. These tests may be applied also to the *vapour* of the acid; they may be occasionally serviceable to corroborate the results.

Objections to the tests.—Nitrate of silver it is well known gives a white precipitate with numerous acids; but if the properties of the precipitate especially those marked under *e* and *h*, be procured, there is at once an end to the objection: *a volatile acid containing cyanogen* must have been present in the liquid. The production of Prussian blue from the liquid, in the manner described, is free from any chemical objection. There is no acid or substance known to chemists that will, under similar circumstances, produce the same results. The sulphur-test is also free from any objection, the sulphocyanate of iron being well characterized by its blood-red colour, its solubility in a solution of corrosive sublimate, and by other chemical properties.

When the tests are applied to the detection of the *vapour*, they are absolutely free from any chemical objection. There is no other acid, nor any volatile compound of cyanogen, which in any respect resembles prussic acid, that can produce similar results. The tests applied to the *vapour* are therefore *conclusive*. There can be no pretence for omitting this part of the process in any medico-legal analysis.

Delicacy of the tests.—The silver test. A standard solution was made, in which each grain of liquid contained the 4400th part of a grain of anhydrous prussic acid. One grain of this very diluted liquid placed on a plate of glass, gave a decided milkiness with nitrate of silver; but when the same quantity was mixed with sixty grains of water, nitrate of silver had no perceptible effect; showing the powerful influence of dilution on the action of a test: the 4400th of a grain of prussic acid being in this instance diffused through 264000 parts of water. The 1460th of a grain in sixty grains of water (dilution 87600) gave, with the nitrate of silver, a milkiness, but no precipitate. With the 440th gr. (dilution 26400) the milkiness produced by the test increased, and the white cyanide became flocculent; and with the 220th gr. (dilution 13200) the test gave a decided precipitate. Strong nitric acid was added to the liquid, but the precipitate was not entirely dissolved even on boiling.

Thus, then, in a minimum of water, the silver test will produce a perceptible effect with less than the 4000th part of a grain of anhydrous prussic acid; but it only begins to give a decided precipitate with the 220th gr. in about 13000 parts of water. Even then, the principal properties of the precipitate cannot be brought out; because, from so small a quantity of cyanide of silver as that which was here formed, it would be impossible to produce cyanogen in sufficient quantity for combustion. Practically, therefore, the limit for the silver-test in detecting prussic acid must be at that point at which it ceases to produce a sufficient quantity of the cyanide of silver either for the production of cyanogen by heat (*e*,) or of sulphyocyanate of ammonia by muriatic acid (*h*.) No medical jurist should rely upon the production of a faint white precipitate in a liquid, as a proof of the presence of this poison.

If the dry cyanide be heated in a short tube of about the eighth of an inch diameter, from one-third to one-fourth (Orfila says three-quarters) of a grain of the precipitate, will be sufficient to produce cyanogen for combustion. This is equivalent to about from one-twentieth to one-fifteenth of a grain of anhydrous prussic acid. A minute flame of cyanogen may in a careful experiment be obtained from so small a quantity as the *tenth* of a grain of cyanide of silver; and unless we obtain that quantity of dried precipitate, we cannot be sure, from the action of this test, that we are operating on prussic acid. The tenth of a grain of cyanide of silver is equal to about the fiftieth of a grain of anhydrous prussic acid—a quantity contained in about *two drops* of the Pharmacopœial acid, or in less than *one drop* of Scheele's acid. In order that the silver test should act for the production of cyanogen, there must, therefore, be present at least the 50th of a grain of anhydrous prussic acid; and if that quantity be much diluted, the liquid must be distilled before the test is added. When the quantity of cyanide of silver is so small that no cyanogen for combustion can be procured from it, its nature may still be determinedby wetting it with muriatic acid, and applying the sulphur-test (see ante, p. 547.)

The iron test.—It is well known to all chemists that prussic acid will not re-act upon either oxide of iron separately to form Prussian blue; but when the oxides are mixed, as they always are in the common green sulphate of iron, the change takes place, and Prussian blue is immediately seen on removing the surplus oxides by a diluted acid. The colour produced by this test is peculiar, and easily distinguishable, even in small quantity, provided we are operating on a *colourless* liquid. It does not, however, appear to be so delicate a test as the nitrate of silver; although, if we regard the necessity for the production of a sufficient quantity of cyanide of silver to evolve cyanogen by heat, there is no great difference. In the use of the iron-test, the collection of the precipitate, and the determination of its chemical properties, are unnecessary: the *colour* alone is sufficient to identify it. The following experiments were performed, in order to determine its delicacy:— One drop of acid (= 1-100th gr. anhydrous acid) was mixed with about 6000 parts of water. The test was applied in the usual way, but there was scarcely any perceptible change. A faint blue tinge appeared, which would certainly not have been noticed, had not attention been specially directed to it. With a similar quantity of acid, thus diluted, the nitrate of silver gave a faint precipitate of cyanide of silver. A solution was now made containing the 50th of a grain of anhydrous acid in 3000 parts of water. The iron-test produced at first only a faint *green* tinge in the liquid; but in the course of twenty-four hours this subsided into a visible deposit of Prussian blue, the liquid above being left perfectly clear. From several experiments this appeared to be the limit, both in regard to quantity and dilution, at which the oxides of iron began to indicate the presence of prussic acid. The quantity here detected by the test is equal to that already described as necessary for the production of a sufficient quantity of cyanide for the evolution of cyanogen; *i. e.* two drops of the Pharmacopœial acid, or less than one drop of Scheele's acid; but there is this difference in favour of the iron test, that it is unnecessary to collect and examine the precipitate, while this is absolutely necessary in the case of the silver test. With the 33d part of a grain of anhydrous acid in 1980 parts of water, the action of the iron-test was perfect and satisfactory. A blueish-green tint was produced in the liquid (a result which is always observed when the quantity of Prussian blue is small and finely divided,) and in about an hour a distinct precipitate of Prussian blue had subsided. Had the poison been even more largely diluted, the test would have detected it. By increasing the quantity of anhydrous acid, *i. e.* when the 20th of a grain was diffused in 1300 parts of water, a dense precipitate of Prussian blue was formed.

The sulphur test.—In a set of comparative experiments performed with this test, the following results were obtained. The prussic acid employed on this occasion, was found to contain 1·44 grains per cent. of anhydrous acid. Two drops of the acid (= ·028 gr., or about 1-36th of a grain of anhydrous acid) were placed in a watch-glass, and gently heated with a drop of hydrosulphuret of ammonia. When the liquid had entirely lost its *yellow colour*, an opaque film appeared on the glass; and, on adding one drop of a solution of persulphate of iron, an intense *blood-red colour* was immediately produced. The colour was entirely destroyed, and the liquid became colourless, on the addition of two or three drops of a solution of bichloride of mercury. A thin film of sulphur, deposited in the experiment, might have been easily separated by water, but it did not in the least degree interfere with the chemical reaction upon which the operation of the test depended. Two drops of this diluted acid were diffused in 220 grains of distilled water; and each grain of the mixture would therefore contain no more than 1-7860th (220 ÷ ·028) of a grain of anhydrous acid. In order to compare the effect of the new with the old tests, ten minims of this very diluted acid were tried with the common Prussian blue test. The liquid acquired a yellowish tint; but after several hours there was no perceptible deposit of Prussian blue. Thus the iron-test failed entirely to detect the 1-786th of a grain of prussic acid in ten minims of water. It required one drachm and a half of this diluted acid (= 1-87th grain) to produce after twenty minutes, a perceptible deposit of Prussian blue of a greenish colour. A like quantity (ten minims) was taken, and a solution of nitrate of silver added to it. There was only a faint opalescence, but no deposit of cyanide of silver. The silver test therefore failed to detect the 1-786th of a grain of the acid; for the production of a faint opalescence could not be regarded as indicative of the presence of the poison. The action of the test became apparent with one drachm of this diluted acid (= 1-131 part of a grain,) but still there was no separable deposit of cyanide of silver. In this highly diluted condition there was not the least perceptible *odour* of the poison. Mr. Hicks states, that by means of the silver test he detected prussic acid in a mixture containing one minim of Scheele's acid to thirty-two ounces (14,080 parts of water.) The odour and all other tests failed to indicate it. (Med. Gaz. xxxvi. 631.) Two minims of the diluted acid were now placed in a watch-glass with one minim of diluted hydrosulphuret of ammonia, and gently evaporated to dryness. A drop of the persulphate of iron added to the residue, gave immediately a reddish tint indicative of the presence of sulphocyanic acid, or a sulphocyanate. The quantity of anhydrous acid which was here clearly detected by the test, was not more than 1-3930th of a grain! (7860 ÷ 2.) It is evident therefore from these results, that the sulphur-test is capable of indicating the presence of prussic acid *when no odour can be perceived*, and *when the iron and silver tests fail to act with any degree of certainty.* The *rapid* action of the sulphur test may be judged of by the following facts: twenty drops of prussic acid, so diluted as to have a strength in anhydrous acid of only 0·48 per cent., therefore rather less than one-fourth the strength of the London Pharmacopœial acid, were placed in a watch glass. On inverting over it another watch-glass containing a drop of nitrate of silver, it was whitened immediately; a drop of hydrosulphuret of ammonia was then substituted. By two separate trials, sulphocyanate was found to have been formed in *ten seconds*, and the effect was very strong in thirty seconds; the quantity of anhydrous prussic acid here detected was ·096 gr., or about 1-12th of a grain. It would be in general advisable to expose the hydrosulphuret to the vapour for one or two minutes, and when the acid is very diluted, for from ten minutes to half an hour. The following experiment will, however, show that the absorption is extremely rapid, even when the acid is much diluted. Three drops of the prussic acid above employed, were diffused through two drachms of distilled water: no odour whatever could be perceived. Ten drops of this mixture were placed in a watch-glass, and nitrate of silver was speedily whitened by the vapour. The effect with hydrosulphuret of ammonia was distinctly procured in five minutes; the total quantity of anhydrous acid present in the ten drops of liquid, did not exceed the 1-473rd part of a grain! The cause of this highly delicate reaction of the sulphur test is, that we obtain the residue in a solid, and therefore in a most concentrated state. There is no water to dilute or destroy the intense blood-red colour characteristic of the sulphocyanate of iron. Hence the chemical changes are most perfectly brought out on the smallest visible quantity of residue. The only precaution required is, not to add too much of the persulphate of iron; a fractional part of a drop will commonly suffice. The salt of iron should be used in a concentrated state, as neutral as possible, and not added until the liquid is colourless. (See Med. Gaz. xxxix. 765.)

DETECTION OF PRUSSIC ACID IN ORGANIC LIQUIDS.

Any organic liquid suspected to contain prussic acid—the matters first vomited, or the contents of the stomach after death, may, under the limitations already mentioned (page 521, ante,) have an odour of the poison perceptible to one or more individuals. If the liquid has no odour of prussic acid, or an odour of some strongly-smelling substance, *e. g.* essential oil, still the poison may be present, and it may be detected, if not as a *liquid*, by the ordinary process of distillation—at least by its *vapour*. With respect to the evidence from odour no better rule can be given than that contained in the observation made by Mr. Baron Parke at the trial of *Tawell*. "The odour is a proof of the presence of the poison, but the absence of odour is no proof of its absence." Of the two processes to be pursued, that which relates to the detection of the *vapour* is the more certain, and open to the least objection. It should always be tried before resorting to distillation, because then no plausible objection can be raised on the ground that prussic acid might have been generated from a decomposition of animal matter during the process. If the poison be clearly and unequivocally detected by its vapour, there is no necessity for resorting to distillation, except for the purpose of determining the proportion of prussic acid present.

Detection by vapour without distillation.—The organic liquid may be placed in a wide-mouthed bottle, to which a watch-glass has been previously fitted as a cover. The capacity of the bottle may be such as to allow the surface of the liquid to be within one or two inches of the concave surface of the watch-glass. The solution of nitrate of silver is then used as a trial-test in the way already described (page 547, ante.) If the 1-200th part of a grain of prussic acid be present and not too largely diluted, it will be detected (at a temperature of 60°) by the drop of nitrate of silver being converted into an opaque white film of cyanide of silver, the chemical change commencing at the margin. This, if in very small quantity, may be found to be soluble in concentrated nitric acid: the solution is aided by heat. It might be *objected*, that muriatic acid contained in the stomach would act on the nitrate of silver in a similar way, but the results of experiments do not bear out this objection. Having mixed one drachm of pure fuming muriatic acid with eight ounces of porter, and placed a watch-glass three inches above the level of the liquid, there was scarcely any film formed on the nitrate after half an hour; while *two-thirds of a grain* of anhydrous prussic acid, mixed with *eight ounces* of porter, placed under precisely similar circumstances, gave a well defined deposit of cyanide of silver in a quarter of an hour! The proportion of muriatic acid was here much larger than that which exists naturally in the gastric secretions, so that except in cases of poisoning by muriatic acid, the objection could not hold. Besides, chloride of silver is insoluble in *boiling* nitric acid, and changes rapidly when exposed to light; the cyanide is soluble, and is but little affected by light. If bromine and iodine be present in a free state, their vapours will produce a film with the salt of silver, but it is of a yellowish-white colour, and the presence of these bodies would be at once indicated by their peculiar odour. This, after all, is merely employed as a trial-test. If the effect take place rapidly with the nitrate of silver, it will also answer speedily with the sulphur and iron tests. (For the mode of applying these see page 548, ante.) In the case communicated to me by Dr. Geoghegan (ante, page 534,) the contents of the stomach, after three days' loose exposure, gave perfectly satisfactory results with the sulphur test. This test will act unequivocally where the iron-test fails. Experiments already related show that it is as delicate in its reaction for this poison, as Reinsch's process is for the detection of arsenic; for it will indicate its presence when the quantity of anhydrous acid is no more than 1-4000th part of a grain! The iron-test also gives satisfactory results: thus I obtained a perceptible quantity of Prussian blue from the vapour of *two-thirds of a grain* of anhydrous acid diffused through eight ounces of London porter. In an organic liquid much diluted, the action of these tests is very slow. The quantity of prussic acid thus detected (like arsenic by Marsh's process) is of course exceedingly small; but on this question I must refer to what has been said at page 117. The analyst may easily procure, by successive experiments, as much Prussian blue, or red sulphocyanate of iron, as will satisfy him that the poison is really present: more than this is unnecessary.

Objections.—With regard to a process so delicate as that of the application of the *sulphur-test*, it may be a fair matter of inquiry whether there are any objections to the results obtained in the absorption of the vapour of organic liquids by the hydrosulphuret of ammonia. It is obvious that, for the effects to follow, there must be some compound of cyanogen capable of uniting with sulphur to form sulphocyanic acid, and capable of being evolved from a liquid at common temperatures in a few seconds. So far as I am aware, no such body exists. If it did, and these results were obtained, they would still furnish a proof of its containing a powerful poison—cyanogen. Thus when prussic acid was mixed with bromine and iodine, the action of the silver-test was obscured, while the sulphur-test acted perfectly; but these compounds, the bromide and iodide of cyanogen, are well characterized by other properties, and, owing to their peculiar and powerful odour, they can never be confounded with hydrocyanic acid. The preliminary test of nitrate of silver will besides be of service in certain cases which might present some apparently ambiguous results. The *sulphocyanic acid* exists in the saliva; but as it is combined with a base, it cannot be volatilized at common temperature so as to mix with the hydrosulphuret, and lead to error. As white mustard contains a principle (*sulphosinapisin*) capable of being converted to sulphocyanic acid, a quantity of the seed was bruised, placed in a watch-glass, and exposed to hydrosulphuret of ammonia. The pulp immediately acquired a yellowish tint from the vapour of the ammonia, but after a quarter of an hour's exposure, the hydrosulphuret left no sulphocyanate on evaporation. With respect to the *iron-test*, it is open to no objection, since Prussian blue cannot be procured, under the same circumstances, from any other vapour except that of prussic acid.

Detection after long periods.—Provided the organic liquid has not been too frequently exposed to air, as under circumstances which would allow of evaporation, the poison may be discovered after very long periods. I have thus detected it, when in small quantity, in porter after the lapse of twelve months. Even the admixture with animal substances in a state of *decomposition*, does not prevent its detection by this process. As a proof of this, the following experiment may be mentioned. The putrified contents of the stomach of a female, who had died under a suspicion of irritant poisoning about three weeks before, were taken for the purpose of trial. The odour was highly offensive; acetate of lead was immediately blackened by the vapour, and the trial test of silver was also blackened; consequently in this case it could not be used for the preliminary detection of prussic acid. To *one drachm* of this offensive liquid, *one drop* of the acid already employed (= ·014 or 1-71st of a grain of anhydrous prussic acid) was added, and a watch-glass, with hydrosulphuret of ammonia, was placed over it. In a few minutes the presence of prussic acid was most satisfactorily detected. To an equal portion of decomposed vomited matter, consisting of blood, mucus, bile, and other substances, a similar quantity of the acid was added; and when the hydrosulphuret was exposed to the vapour, the results obtained were equally satisfactory. Nitrate of silver, for the reason above stated, could not be safely employed in this case. Thus, then, the actual presence of sulphuretted hydrogen in the contents of the stomach, from decomposition, does not interfere with the action of the *sulphur-* while it entirely destroys that of the *silver-* and *iron-tests.*

Detection by distillation.—This process was originally suggested by Lassaigne. The organic liquid may be distilled in a water bath, at 212°, and about one-sixth or one-eighth of the contents of the retort collected in a receiver kept cool by water. The tests may now be applied to the distilled liquid. If the trial tests indicate that the quantity of poison is small, a solution of nitrate of silver or caustic potash may be placed in the receiver, to fix the acid as it distils over; Prussian blue may then be procured in the way described, or the vapour may be at once absorbed by hydrosulphuret of ammonia in the receiver, and the liquid evaporated to obtain sulphocyanate. Prussic acid has been found in the stomach by distillation, so late as *seven* days after death, although the odour could not be perceived before distillation. (Case of *Ramus*, ante, p. 521.) Orfila is said to have discovered it *eight* days after death in the cases of the Parisian epileptics, but he merely states he perceived an *odour of bitter almonds*, not that he obtained the poison by distillation! (See p. 545.) In a case in which three drachms had been taken, I could neither detect the acid by the odour nor by the most careful distillation, *twelve* days after death. (Guy's H. R., April 1845.) Mr. West states that he was able to detect prussic acid, on distillation, by the odour and the silver and iron tests, *twenty-three* days after death; although no pains had been taken to ensure its preservation, and not more than four-tenths of a grain of anhydrous acid could have originally existed in the contents of the stomach! (Prov. Med. Jour., July 23, 1845.) It is impossible to reconcile these discordant results; but the vapour-tests clearly prove that the acid is always escaping from the organic liquid holding it. If sulphuretted hydrogen or hydrosulphuret of ammonia be present in the liquid, it is liable to be distilled over, and completely obscure the results. There is also another singular source of fallacy brought to light by the discovery of the sulphur test—namely, that if hydrosulphuret of ammonia, or any alkaline sulphuret, were present in the organic liquid, the prussic acid would be lost by conversion to a soluble sulphocyanate. Before proceeding to distillation, therefore, it is advisable to test the liquid for sulphur and ammonia:—the first may be detected by inverting over a portion, a watch-glass containing a drop of the acetate of lead: if sulphur be present in a volatile state, the salt is immediately blackened. The alkalinity of the liquid, and its odour, will indicate the ammonia. It is certain that this distillatory process may fail from various causes, when that applied to the detection of the vapour will succeed. Under the distillatory process, the poison is necessarily diffused through a large quantity of water. I have, nevertheless, found that it was easy to detect and separate by distillation only two-thirds of a grain of anhydrous acid diffused in eight ounces of London porter; but the process by testing the vapour is far more delicate than this. Mr. West detected the vapour of prussic acid in the blood by the use of the nitrate of silver, but could not separate any by distillation. (Prov. Med. Jour., July 1845.) Mr. Hicks informs me that in some of his experiments the distillatory process entirely failed, and that he could only detect the poison by the method above described. On one occasion he mixed thirty drops of Scheele's acid (= 1·12 gr. anhydrous) with half a pint of porter, and poured the mixture into the stomach of a recently dead subject. After *two* days he examined the contents, but neither himself nor several persons present could detect any smell whatever of prussic acid. However, in a quarter of an hour, satisfactory evidence of its presence was procured by the absorption of its vapour by nitrate of silver. (See Medical Gazette, xxxvi. 682.)

Mr. Hicks forwarded to me one-half of the liquid, and my results confirmed his. What a commentary does this experiment furnish on the sophistry allowed to be put forward in defence, as received medical doctrine, at *Tawell's* trial! (p. 544.) This process, like that of the vapour process, is open to the *objection*, that a soluble cyanide (potassium) may produce precisely similar results. This, however, is merely substituting one poison (ejusdem generis) for another. The presence of potassium mignt be determined by neutralizing a portion of the liquid with muriatic acid, and evaporating to obtain chloride of potassium.

Absorption of prussic acid.—There can be no doubt that prussic acid is *absorbed*, and enters into the blood: it thus penetrates all the tissues of the body; and from the great diffusibility of the vapour, the odour may be perceptible in cavities and organs remote from the stomach, even before this viscus has been opened. The presence of the odour, but especially of the poison, in the blood, furnishes strong evidence of poisoning: the odour is only likely to be met with in this liquid in very recent cases. Prussic acid has been detected in the blood and viscera of animals poisoned with it, by the old process of distillation. Krimer some years since discovered it in the blood of an animal which was killed by the poison in thirty-six seconds: the analysis was made by distilling the liquid to dryness. Heller states that he detected it in most of the soft organs, as well as in the blood, by digesting them in a small quantity of potash, and then adding a salt of iron and muriatic acid. (Archiv. für phys. Chem. 1845, i. and ii. 143.) As a proof of its absorption and universal diffusion, the following experiment, performed by Mr. Hicks, may be adduced. He poisoned a cat with twenty minims of Scheele's acid. Eight hours after death the body was examined. A small aperture was made in the chest, and a watch-glass, containing a few drops of nitrate of silver, was accurately fitted over it. In half an hour there was a perceptible white margin, and in an hour the whole of the nitrate had been converted to a white film, proved to be cyanide of silver by its insolubility in cold, and its solubility in boiling nitric acid. The whole of the contents of the chest, placed in a bottle, gave out a vapour which readily produced cyanide of silver and Prussian blue with the respective tests. (Med. Gaz. xxxvi. 632.)

Detection of prussic acid in the tissues.—The poison may be easily detected in the blood, secretions, or any of the soft organs, by placing them in a bottle, and collecting the vapour in the way already described (ante, p. 533.) This will be found to be far more convenient and satisfactory than the process by distillation. In the case of a dog poisoned by prussic acid, Mr. Hicks brought me the stomach after it had been exposed twenty-four hours, and thoroughly washed under a current of water, and yet the poison was readily detected by placing the whole organ in a bottle, and absorbing the vapour by nitrate of silver. This shows how completely the animal tissues are penetrated by prussic acid, and how firmly it is retained by them. The poison has been thus discovered in the blood and the serous exhalation of the chest.

The poison not detected.—The causes of the failure of chemical analysis have been already explained in speaking of the loss of odour. The principal of these during life are absorption and elimination, and after death, evaporation. The distilled liquid of the stomach of a dog poisoned by prussic acid gave clear evidence by all the tests one day, but after loose exposure no trace of the acid could be detected in it the following day. I am not aware of any case in which the poison has been detected for a longer period than that described by Mr. West (ante, p. 555)—twenty-three days. All traces of the acid are generally lost in about one-half of this period. A question may here arise, whether it might not be fixed by alkalies, such as ammonia, from putrefaction; but the combination of prussic acid with an alkali does not prevent the continual evolution of the vapour, as the following experiment with the sulphur test will prove. To one drop of prussic acid (1·4 per cent.) two drops of a moderately strong solution of ammonia were added: the liquid was strongly alkaline. Nitrate of silver was rendered opaque by its vapour, and the hydrosulphuret gave the usual evidence of prussic acid in a few minutes. When a very large excess of strong solution of ammonia was added, there was no effect on exposing to the vapour, the nitrate of silver and the hydrosulphuret; but by diluting the liquid, and then acidulating it with diluted sulphuric acid, satisfactory results were obtained with both tests in a few minutes.

Value of evidence from the detection of prussic acid in the stomach and in the tissues.—Is the discovery of prussic acid in the stomach of a person, a proof that death has been caused by it? As a general rule, we should be justified in answering this question in the affirmative: as less than a grain may destroy life. It has been suggested that the poison might be introduced or spontaneously generated after death; but if such improbable speculations as these are to restrict medical evidence, no certainty can ever be obtained. We do not here meet with the objection which applies to most other poisons, that the patient may have been cut off by disease supervening after it has been taken; since if the poison operate fatally at all, it is in the course of a few minutes. Latent diseases of the heart and brain might, it is true, by a coincidence cut short life; the circumstances of the case may, however, be such as to remove any supposition of this kind. The detection of the acid in the tissues, in a remote organ, or in the blood, would render it absolutely certain that it had been taken into the body during life. When two or more poisons are found in the stomach, and one is prussic acid, there can be no reason to hesitate in assigning death to the latter. In a case which occurred in 1837-8, prussic acid and arsenic were found in the stomach after death. In another, communicated to me by Mr. Clarkson, formerly a pupil at Guy's Hospital, the mixture taken by the deceased consisted of brandy, opium, arsenic, and prussic acid. The witness must therefore be prepared for these cases of compound poisoning, and the questions arising out of them. Prussic acid, according to the quantity discovered, must not only be regarded as the cause of death, but as the poison last taken, unless there be reason to suppose that the whole of the poisons were swallowed in one mixture.

Spontaneous production of prussic acid.—If the analyst confined his processes to the detection of free prussic acid in the stomach, it might be objected to his results that the poison was spontaneously generated. If he detected it only *after* distillation, it might be alleged that the heat employed reacted on organic matter and produced it. Supposing that the presence of the poison has been determined in the blood and remote viscera by the *vapour* tests, it is obvious that the objection at once falls to the ground. This part of the evidence may be neglected, or it may fail: hence it is necessary to consider whether prussic acid can be spontaneously generated under any circumstances, in organic liquids or in the body, within a few days after death.

That this acid is a product in various chemical processes is undoubted; thus it appears to be formed at a high temperature by the reaction of nitric acid on alcohol (see ante, p. 519,) as well as in other cases. No one has yet suggested that there is *normal prussic acid* in the body: hence the whole question turns upon the chemical possibility of its spontaneous generation from articles of food or other substances which may be contained in the stomach. That the acid should be thus produced without the aid of heat, has not, so far as I am aware, been asserted; and if it were liable to be produced by heat during distillation, it is obvious that those who are engaged in medico-legal researches would be continually meeting with it as a product. There is nothing to bear out such an hypothesis in the works of the most eminent writers on toxicology. If the organic liquid contained any of the kernels of certain fruits, the pulp of the black cherry, or substances which are known to furnish prussic acid by mere contact with water, the poison would be undoubtedly procured by distillation; but the result is then artificial, and cannot constitute a valid objection to the chemical processes employed. Orfila admits that the secretion of the axillæ may occasionally have an odour of prussic acid; but there are many fallacies connected with a reliance on odour. He also believes it to be possible that prussic acid may be a product of decomposition in the dead body, like acetic acid or ammonia, or, at any rate, that putrefaction may engender some compound which will give with the different tests all the reactions of prussic acid. (Toxicologie, ii. 323.) This is put as a bare possibility from analogy; and as a corrective to the doubt thus thrown on chemical evidence, he says the objection is inadmissible when the individual has died under the symptoms of poisoning by prussic acid, and when the appearances in the body are like those caused by this poison! This, however, would not meet those cases where nothing was known concerning the symptoms. He further states that if the organic liquid is not much decomposed, and only a gentle heat is applied during distillation, the objection cannot hold, because the poison is not obtained under these circumstances. Orfila does not, however, adduce a single fact in support of his views; and the result of the only experiment which he mentions is decidedly against them. He obtained from the distillation of partially decomposed matters a liquid of a fetid odour, *sometimes* precipitable by nitrate of silver, the precipitate being *soluble* in nitric acid. The liquid gave a greenish precipitate with sulphate of iron and potash, which disappeared entirely on adding muriatic acid, without leaving a trace of Prussian blue. (Toxicologie, ii. 313.) It is easy to perceive that there is not here a single chemical character of prussic acid.

M. Bonjean, of Chambery, has recently examined this question. After stating what is a well-known fact, but quite irrelevant to the present question, that organic substances at a high temperature (a red heat) produce cyanogen compounds, he gives the results of some experiments on the distillation of beef with water, and of the pure blood of the ox; also of various mixtures of organic matters and of animal matters, in a state of putrefaction (op. cit. 54;) but in not one instance was prussic acid proved to exist, except when a substance containing it had been added! The results of experiments do not, therefore, bear out this hypothesis; and if analogy is to be substituted for proof in its favour, there is nothing to prevent the admission of the spontaneous production of morphia, strychnia, and other organic poisons! The alleged production of prussic acid by the distillation of organic matter under a temperature of 212°, appears, therefore, to be an unfounded objection. During the last seventeen years, I have distilled the contents of numerous stomachs, in all states of decomposition,—organic liquids, such as porter, milk, gruel, either mouldy or putrid, without in any one instance finding the slightest trace of prussic acid in the distilled liquids, either by the odour or by the usual tests; and it appears to me that until some unequivocal results in support of this hypothesis be forthcoming, a medical witness would be fully justified in rejecting it as an unfounded assumption.

When the stomach contains matters from which pussic acid is known to be chemically producible by simple admixture with water, with or without the application of heat, the case is different. It then becomes a question, whether the quantity of prussic acid which these substances are capable of yielding, bears any proportion to the actual quantity detected. One bitter almond will give a perceptible quantity of Prussian blue, when the pulp is triturated with cold water, and treated with sulphate of iron and potash in the usual way; but the blue colour only becomes visible in the midst of the white pulp, after some hours. Distillation is not necessary for the production of the poison in this case; for the results is easily obtained at a temperature of 60°. It is said to have been found by Dr. Witling as a spontaneous product of the decay of unsound cheese. Dr. Christison states on the authority of Dr. Witling. (756.) In one experiment I examined 230 grains of decayed cheese, by digesting it in a small quantity of distilled water, and gently distilling the mixture. The liquid thus obtained had the odour of decayed cheese, and contained ammonia, but not the slightest trace of prussic acid could be detected in it by the most delicate tests. This experiment was repeated with Stilton and other cheeses in various states of decay; but even the sulphur-test failed to show that any portion of prussic acid was present. Large quantities of decayed cheese are sold to the poor in London, but we never hear of any effects like those caused by prussic acid resulting from its use. I have found such cheese to be of a brown colour, of a highly offensive odour, and possessing an acrid bitter taste. The decayed portion of the better kinds of cheese, in all stages of animal decomposition, and covered with vegetable growths, are, it is well known, eaten by the epicure also without accident. If prussic acid were produced as it is alleged, nothing could prevent its volatilization as rapidly as it was formed, unless it was pretended that the acid was fixed! It appears to me from these facts that Dr. Witling's statement requires corroboration before it can be received. Unsound cheese may act as an irritant (ante, p. 445,) but its poisonous effects are then not due to the presence of prussic acid. (See also Med. Gaz. xxxvi. 328.) The ergot of rye is said to contain prussic acid by one chemist, but this statement has been denied by another: the question, therefore, remains unsettled.

Notwithstanding the entire absence of proof that prussic acid is generated, either at common temperatures or at the heat of distillation (212°,) it is necessary to state that a salt (sulphocyanide of potassium) containing cyanogen, exists in minute traces in the saliva. This may possibly be mixed with the contents of the stomach (page 505, ante,) and yield prussic acid, not on simple distillation but by distillation with an acid. In answer to this it may be stated that if the vapour tests had given the usual results, the objection could not hold, because the sulphocyanic is a fixed acid and in a state of combination, but experiment shows that the objection is wholly unfounded. On distilling a strong solution of the sulphocyanide with sulphuric acid, at a gentle heat, no prussic acid could be procured: the distilled liquid consisted simply of sulphocyanic acid, sulphur, and sulphuretted hydrogen. This hypothetical objection was taken at *Tawell's* trial to the chemical evidence, but as the above facts show, there is not the slightest foundation for it. If it were true, a human stomach would always contain prussic acid from the reaction of the muriatic acid in the gastric secretions on the sulphocyanide contained in the saliva!

QUANTITATIVE ANALYSIS.

It is often a matter of great importance, to ascertain the strength of the prussic acid taken! and it is much more satisfactory to determine this point by chemical processes, than by giving the poison to dogs or rabbits, and noting how long a time it requires for a certain dose to destroy life, or by assuming its strength from its name. In performing this experiment, it is necessary to precipitate a weighed quantity of the acid entirely by solution of nitrate of silver, and wash and dry the precipitate in a water-bath until it no longer loses weight. One hundred grains of cyanide of silver are equivalent to 20·14 grains of anhydrous prussic acid; this is in the proportion of about one-fifth, so that the weight of the dried cyanide, divided by five, gives with sufficient accuracy for common purposes, the quantity of anhydrous prussic acid present. One hundred grains of the *London Pharmacopœial acid* should therefore yield ten grains of cyanide of silver, and of *Scheele's acid* from twenty to twenty-five grains. Before making the calculation it is most important that the cyanide of silver should be thoroughly dried. It holds water very tenaciously, and unless this be *entirely* expelled, a very erroneous opinion may be formed of the strength of the acid examined. It may be also necessary to determine how much of the acid exists in the stomach or in a liquid requiring analysis. The whole, or if large, a fractional part of the liquid, should be distilled, and the clear product treated in a similar way.

CHAPTER XXXIX.

PRUSSIC ACID POISONOUS WHEN COMBINED WITH ALKALIES—CYANIDE OF POTASSIUM—SYMPTOMS AND POST-MORTEM APPEARANCES IN MAN AND ANIMALS—FATAL ACCIDENTS FROM—LOCAL ACTION ON THE SKIN IN GALVANIC GILDING—QUANTITY REQUIRED TO DESTROY LIFE—ITS STRENGTH COMPARED WITH PRUSSIC ACID—TREATMENT—CHEMICAL ANALYSIS—SULPHO- AND FERROCYANIC ACIDS AND THEIR SALTS—ANALYSIS—PRUSSIAN BLUE—CYANIDES OF MERCURY AND SILVER—ANALYSIS—BITTER ALMONDS—POISONOUS PROPERTIES OF—ESSENTIAL OIL—SYMPTOMS AND APPEARANCES CAUSED BY—RETENTION OF CONSCIOUSNESS AND VOLITION—QUANTITY REQUIRED TO DESTROY LIFE—REMISSION OF SYMPTOMS—EFFECTS OF THE VAPOUR—ANALYSIS—BITTER ALMOND-WATER—POISONOUS PROPERTIES OF—STRENGTH OF—ANALYSIS—LAUREL OIL AND WATER—NOYAU, CHERRYRATAFIA—POISONING BY CHERRY AND PEACH-KERNELS—PLANTS CONTAINING PRUSSIC ACID.

General Remarks.—Hydrocyanic acid is as fatal to animal life when combined with alkaline bases, as when it is free. Thus, the same quantity of diluted prussic acid will kill a dog, whether it be given in a pure state, or combined with ammonia or potash (Orfila, ii. 292.) Hence, ammonia cannot be regarded as a chemical antidote in cases of poisoning by prussic acid, but merely as a stimulant. Alkalies have not even the power of fixing the acid, a fact proved by the ready action of the vapour-tests on all the salts of hydrocyanic acid. This question arose in the case of *Reg.* v. *Cronin*, C. C. C. 1847. The prisoner had prescribed aromatic spirits of ammonia with prussic acid, and the question was, whether the poison would become thereby in any degree disarmed of its virulence. The answer was certainly in the negative, as the rapid death of the female proved. When mixed with an alkaline base, such as ammonia, prussic acid is liable to undergo speedy decomposition. It becomes yellow, then brown, and finally, almost black, a thick black sediment being formed in it. This change is, however, only partial: in one specimen thus altered, I found a large quantity of free prussic acid after two years. The change is not observed to take place when the prussic acid bears a small proportion to the base, or vice versâ; nor does it so readily occur when the prussic acid is diluted.

The only saline compound of prussic acid which is of any practical interest as a poison, is the Cyanide of Potassium.

CYANIDE OF POTASSIUM.

This is one of the most formidable poisons known to chemists. It has led to the destruction of life in several instances within the last few years, chiefly owing to its having been administered by mistake for other medicinal preparations.

The **Symptoms** which it produces are similar to those occasioned by prussic acid:—spasmodic respiration, convulsions, with tetanic stiffness of the limbs and trunk. They appear in a few seconds or minutes, and run through their course with great rapidity. Orfila found that four grains and a half dissolved in water, and injected into the pharynx of a dog, produced well-marked symptoms in ten seconds; and there was convulsive respiration, followed by apparent death in five minutes. Vomiting of a liquid smelling strongly of prussic acid took place, and the animal died in eleven minutes. M. Bonjean gave to a rabbit 8-10ths of a grain of cyanide of potassium dissolved in a small quantity of water. The animal had a violent convulsive fit, and died before it could completely swallow the poison. This gentleman found that animals to which this poison was given invariably had convulsions, tetanic spasms, and abdominal respiration. When administered with some kinds of white wine containing iron, he found that it gave a blueish tinge to the mucous membrane of the alimentary canal. (Faits Chimiques rel. à l'Emp. par l'Acide Prussique, 1843, p. 30.) Bischoff states that a titmouse was killed in half a minute by 1-18th of a grain of the cyanide placed on the tongue, and that a guinea-pig died in violent convulsions in a few minutes from a grain and a half placed on the tongue. (Canstatt's Jahresbericht, 1844. B. v. p. 292.)

Post-mortem appearances.—The great rapidity of death renders it unlikely that any well-marked appearances should be met with. In animals, there has been in the body, when examined recently after death, an odour of prussic acid: this has been also observed in the brain. With this there was an ecchymosed condition of the mucous membrane of the stomach, and congestion of the cerebral vessels with dark-coloured blood. (See Ann. d'Hyg. 1843, i. 409.)

The Cyanide of Potassium is much used on the continent as a medicine, and it has lately occasioned the death of a person at St. Malo, under the following circumstances. A physician prescribed for the deceased, rather more than one drachm of the cyanide in two ounces and a half of orange-flower water and syrup; and of this mixture, three table-spoonfuls were to be taken daily. It seems that only one table-spoonful was taken, and the patient died in three quarters of an hour. None of the poison was found in the stomach; but a portion of the mixture from which the first dose had been taken was examined, and found to contain the cyanide of potassium. A criminal procedure was instituted against the physician, and he was fined and imprisoned. MM. Malaguti, Sarzeau, and Guyot, who gave evidence on he occasion, stated that they found no trace of the poison in the body,—that the cyanide was pure, and only one table-spoonful was missing from the bottle. They further stated, that a dog was killed in a few minutes after taking less than *three grains* of the cyanide in solution, and that the largest medicinal (?) dose to a human being was five-sixths of a grain. The mixture in this case, contained about three grains of the cyanide in one drachm: therefore had teaspoonfuls been taken by the deceased, the quantity would have been quite sufficient to destroy life. The medicine had evidently been prescribed by a person totally ignorant of its poisonous properties. (Ann. d'Hyg. 1843, i. 413.) Another case occurred at Breslau in January 1842, in which a man, aged thirty, died in a *quarter of an hour* after taking a dose of mixture which had been prescribed for him by his medical attendant, under all the symptoms of poisoning by prussic acid. (Henke's Zeitchrift der S. A., 1843, p. 7.) The mistake here arose from those unfortunate changes periodically made in the nomenclature of pharmacopœial compounds, which constitute a matter of regret among ourselves; for such a practice takes away all certainty from the art of prescribing, and leaves the life of the patient and the character of the practitioner in the hands of a druggist, who may be ignorant of the properties of the medicine which he dispenses. It appears that until lately the yellow ferrocyanate of potash was known in the Prussian Pharmacopœia under the short name of "kali hydrocyanicum," just as it was formerly called, in English, prussiate of potash, and is now termed ferrocyanide of potassium,—an objectionable alteration from the term ferrocyanate, because many dispensing druggists might confound the *ferro*cyanide with the cyanide, and dispense the poison for the innocent substance. Of late years, in the Prussian Pharmacopœia, the cyanide of potassium has received the name of "cyanetum kalicum," or, improperly, "kali hydrocyanicum." *Fifteen grains* of "kali hydrocyanicum" in a dose, were prescribed by the physician for his patient; he intended to order the ferrocyanate of potash. Instead of this salt, however, cyanide of potassium was sent, and the patient took the draught and died in a quarter of an hour. On inspection, there was no particular odour: but the poison was detected in the contents of the large intestines. The party who dispensed the medicine, was, in this instance, undoubtedly to blame; for it appears that he entertained some doubt about the largeness of the dose, and he ought to have known that a dose of such a compound, could not be taken by a human being without certainly destroying life.

Two years afterwards (January 1844,) a similar accident occurred in Germany, by which the patient was killed, and the physician had a narrow escape of his life. Two drachms of *kali hydrocyanicum* were ordered, in a prescription, with two drachms of sugar dissolved in two ounces of camomile-water; a dessert-spoonful to be taken every quarter of an hour. Cyanide of potassium was dispensed instead of the ferrocyanide—the salt intended! The patient, an adult, took a dose (about 100 drops,) and the operation of the poison became manifest during the act of swallowing. There was a tendency to vomit, and an immediate loss of consciousness: death took place in an hour. The quantity of cyanide here taken was not less than from thirteen to fifteen grains, equivalent to more than five grains of anhydrous, or 100 drops of Scheele's prussic acid! The physician who prescribed the medicine, was sent for while the patient was still suffering from its effects; and in order to show that he had prescribed an innocent mixture, he put about a teaspoonful of it into his mouth, and swallowed three-fourths. The remainder he spat out, as it gave him an astringent or constricting sensation in his throat, like that caused by alum or green vitriol. He immediately felt severe pain in the back of the head, there was inability to stand, indistinct vision, nausea, a rushing sound in the ears, loss of consciousness, and without complaining of any well-defined pain, he felt that he had lost the power to make a deep inspiration. The loss of sense was as rapid as in ordinary syncope. When an effort was made to swallow some milk, there was a strong sense of choking, followed by copious vomiting. For more than an hour he could not stand upright. Vertigo, weight in the head, and constriction in the throat, continued for many hours. He passed a restless night; but the next day, with the exception of feeling a general relaxation and weakness, he had recovered, and was enabled to assist at the post-mortem examination of his patient!

The body of the patient was inspected two days after death: there was no remarkable odour;—muscles stiff and rigid; the face, and fore part of the trunk, pale; the back part livid, except those portions which had sustained pressure. The fingers and toes were convulsively bent inwards, the nails blue, eyelids half-closed, lips pale, the sinuses and cerebral vessels filled with blueish-red (blaurothem) blood. On making a section of the cerebellum and spinal marrow, bloody points were observed. There was infiltration of the lungs posteriorly, and on cutting into them, a strong odour of bitter almonds was perceived. A yellowish mucus was found in the stomach, which yielded on analysis cyanide of potassium. The mucous membrane was reddened near the pylorus. The poison was not detected in any part of the body except the contents of the stomach and intestines. (Casper's Wochenchrift, Oct. 4, 1845, 657.)

Two circumstances are remarkable in this history—1, that the patient should have survived the dose so long as an hour, and 2, that the physician should have recovered. It is impossible to suppose that he could have swallowed so much of the solution as is alleged, for this would have amounted to at least five grains of cyanide of potassium, and vomiting was not immediate. This case contains a serious caution in reference to a remark made elsewhere, (ante, p. 48,) upon prescribers swallowing their own medicine, to prove that no poison is present!

Local action.—Cyanide of Potassium possesses a *local* action. A patient was directed to use as an enema a solution of rather less than *five grains* (4·6 gr.) of the cyanide dissolved in six ounces and a half of water. He was seized soon afterwards with convulsions, palpitation, slow and difficult respiration, coldness of surface, dilatation of pupils, and fixedness of the eyes. He died in an hour. (Ann. d'Hyg. 1843, i. 412.) It appears that thirty-six hours before, he had used a similar enema without injury, but the probability is, that the cyanide then employed was not pure.

Dr. Chanet has recently directed the attention of the profession to the local action of this poison on the hands of the workmen engaged in the arts of electro-gilding and silvering. The hands of these men are almost always covered with ulcers on the dorsal surface. The skin about the joints is fissured, and an oozing of blood often takes place. The nail with its root participates in the inflammation. The workmen informed him, that on dipping their arms into the bath for a few seconds, the whole of the skin became reddened. The ulceration of the soft parts continued even to the bone, producing great pain and broken rest. (Gaz. des Hôp. 24 Juil. 1847, 374.) The strong alkalinity of the solution would explain some of these effects, for the solution readily dissolves the cuticle, and exposes the true skin. The excoriations produced may, however, lead to the absorption of the poison, and to all the effects of chronic poisoning by prussic acid. Facts are wanting to show how far the lives of the workmen are liable to be destroyed by this newly-invented process. The poison acts upon them not merely locally, but by the vapour, the respiration of which they cannot avoid. (See ante, p. 519.)

The cases just related show that the cyanide of potassium is a most powerful poison, and that it destroys life with great rapidity.

Quantity required to destroy life.—Two grains and a half (2·44 gr.) of the pure salt are equivalent to one grain of anhydrous prussic, or fifty minims of the London Pharmacopœial acid. Hence the cyanide may be regarded as a solid compound of hydrocyanic acid containing of this poison in its most concentrated form no less than 39·3 per cent. by weight!

A dose of from three to five grains of the pure salt may, therefore, easily prove fatal. From a case just related, it would appear that a dose of less than five grains has actually destroyed life.

The energy of the cyanide of potassium as a poison depends, in some measure, on its mode of preparation. Some specimens are so impure as to consist almost entirely of carbonate of potash, from which the cyanide may be separated by its ready solubility in weak alcohol, (see Ann. d'Hyg. 1843, p. 404, in which this subject is fully investigated by Orfila.) Other specimens contain a large quantity of cyanate of potash. These impurities will of course affect its strength. An opinion formerly prevailed, that the poisonous properties of this salt were destroyed under two circumstances:—1, by exposure to air, in which case it is transformed to carbonate of potash: and, 2, by its being heated, in solution, to the boiling point. In neither instance, however, does the salt easily lose its poisonous properties. Orfila found that some which had deliquesced, by exposure to air for a fortnight, still acted as a poison; and the conversion of the salt, at 212°, into ammonia and formate of potash, takes place so slowly, under the most favourable circumstances, as not to interfere with this poisonous action. This substance does not therefore become innocuous, as it was formerly alleged, by solution in *hot* water. I have found by experiment that the ebullition of a solution, continued for a quarter of an hour, produced no sensible quantity of formate of potash.

The cyanide is not used medicinally in England. The *medicinal dose* is estimated at from one-eighth to one-fourth of a grain, but as the salt is of uncertain composition, it is a most dangerous substance to employ. From its great solvent powers on the metals it is very extensively employed in the arts of electro-gilding and plating, and may, therefore, easily give rise to accidents. The solution is improperly kept exposed, and is constantly evolving hydrocyanic acid in vapour.

M. Bonjean has ascertained that this poison is entirely lost by decomposition. After forty days he was unable to detect it by the odour or tests in the stomach of the rabbit which had been killed by 8-10ths of a grain, and into which a like quantity had been introduced soon after death. (Op. cit. 33.)

TREATMENT.—The symptoms occur with such rapidity and violence, that there is scarcely time to apply treatment. The administration of a weak solution of green sulphate of iron would have the effect of decomposing the poison, and converting it to Prussian blue. Cold affusion and the other remedies used in poisoning by prussic acid should be also applied.

CHEMICAL ANALYSIS.—When pure, it appears as a white crystallized salt, or as a chalky looking mass. It has an acrid alkaline bitter taste: it is without any odour until put into water, or until air and moisture have free access to it; it then has the well-marked odour of prussic acid. It is deliquescent, and exceedingly soluble in water: the solution, when pure, is colourless, and has a strong alkaline reaction, a soapy feel, and a powerful hydrocyanic odour. It is not very soluble in pure and strong alcohol. 1. It is decomposed by all acids, and prussic acid is set free. 2. It is precipitated by tartaric acid, and chloride of platina. 3. It gives a white precipitate with nitrate of silver, which, when dried and heated, possesses all the properties of cyanide of silver, (ante, p. 547.) This precipitate is easily redissolved by a slight excess of the solution of cyanide of potassium. 4. If a solution of green sulphate of iron be added to it, and afterwards, diluted muriatic acid, Prussian blue will result, indicating thereby the true nature of the salt. 5. A single grain of this salt moistened with water in a watch-glass, gives a well-marked reaction, by its vapour, with the silver and sulphur tests, (see ante, p. 548.) In *Organic liquids*, the vapour-tests for prussic acid should be applied. If the liquid be rendered slightly acid by diluted sulphuric acid, the effect will be expedited. By distilling the organic liquid with sulphuric acid, prussic acid is obtained in the receiver, and sulphate of potash may be procured by incinerating the residue left in the retort. Advantage may be taken of its insolubility in pure alcohol, to separate from it some organic principles.

SULPHO- AND FERRO-CYANIC ACIDS.

These acids, either free or combined with alkalies, are said not to be poisonous; but further experiments are required to determine to what extent they may be noxious to man. A singular case, in which sulpho-cyanic acid was alleged to have been the cause of death, will be found reported in the Brit. and For. Med. Rev., July 1839. A man wishing to destroy himself, swallowed a liquid which he had obtained by distilling strong sulphuric acid with ferrocyanide of potassium. He was found dead in his room, and twenty-four hours afterwards, the body was examined. The stomach was not inflamed, but part of its mucous surface was softened, and of a brownish-black colour. There was no odour of prussic acid. Some doubt being entertained, as to what the products of such a distillation might be, experiments were performed; but the results obtained by the different experimentalists did not agree. In repeating the distillation, I have found that prussic acid in large, and sulpho-cyanic acid in small quantity, were procured; and it is highly probable that death was really caused by prussic acid, which may have been the case, although no odour was perceptible. The blackened state of the stomach was probably due to some strong sulphuric acid being mixed with it.

Sulphocyanic acid and sulphocyanide of potassium have been found, in moderately large doses, to cause the death of animals, but experimentalists generally agree in assigning very weak powers to ferro-cyanic acid, and its combinations with alkaline bases. Bischoff found that five grains produced tremors in a small rabbit. These passed off; the animal ate its food readily, but died in five days. On a charge of poisoning with the ferro-cyanide of potassium, which occurred a few years since in Germany, the medical witnesses were asked whether it was a poison. They could not answer the question, but said it would undergo a decomposition in the stomach which would render it inert! In this instance there was no proof that the salt had even been swallowed; and the sudden death of the woman appeared to be due to hydrothorax. (Canstatt's Jahresbericht, 1844, B. v. s. 291.) Any acids in the stomach would tend to decompose it, and set free prussic acid; but this change has but little tendency to take place at the temperature of the body (98°.) Further experiments are required to determine the properties of this compound. According to Schubarth, it is not poisonous to man or animals in drachm-doses. It was formerly supposed to contain prussic acid, and that it was therefore highly deleterious. It is now known, however, that prussic acid results from a reaction of its elements, and that it has no independent existence in the salt.

ANALYSIS—SULPHOCYANIDE OF POTASSIUM is a white crystallizable salt, very soluble in water, and forming with it a colourless neutral solution. The tests which may be employed are—1. A *Persalt of iron.* If persulphate of iron be added to a solution of this salt, even when much diluted and in very small proportion, it immediately produces a deep blood-red colour. The colour is destroyed, and a milky-white precipitate is thrown down on the addition of a solution of corrosive sublimate. 2. *Iodic acid.* When added to the solution, iodine is set free, indicated by the blue colour produced on the addition of starch.

FERROCYANIDE OF POTASSIUM.—This is a well known yellow salt, crystallizing in square plates, which are somewhat tough. It easily dissolves in water, forming a neutral yellow solution. A *Persalt of iron* gives with it, even when considerably diluted, a deep blue precipitate, (Prussian blue.) When the powder is warmed with diluted sulphuric acid, prussic acid is set free. This may be procured by distillation, or if the salt be in small quantity, (one grain) it may be proved to exist by the silver and sulphur tests for prussic acid applied to the vapour.

CYANIDE OF IRON. PRUSSIAN BLUE.

This substance does not appear to possess any poisonous properties. It is said to be much employed, when mixed with some yellow colouring matter, to give a green colour to factitious tea-leaves. In a seizure which was lately made of some spurious tea, a question was put by the magistrate—whether Prussian blue was a poison. One of the parties, who gave evidence, is reported to have said that it was a decided poison: that it consisted of iron, nitrogen and carbon, and was strongly impregnated with prussic acid! This evidence appears to have been received without any comment.

Chemical Analysis.—Prussian blue is a tasteless powder of a deep blue colour, insoluble in water, alcohol, and the diluted acids. It may be identified by the following characters. 1. When heated in the air it turns brown and becomes incandescent. Indigo, under the same circumstances, is in great part volatilized in the form of a rich purple vapour. 2. If warmed with a few drops of caustic potash, oxide of iron is precipitated, and ferrocyanide of potassium is formed. When this liquid, neutralized, is added to a solution of sulphate of copper a deep claret red precipitate falls down. (See ante, p. 377.) 3. Its most remarkable property, and that by which it is known from all other blue compounds, is that it forms a dirty white solution when boiled with concentrated sulphuric acid; and Prussian blue is again precipitated when this solution is added to water. 4. Boiled with peroxide of mercury it yields on filtration and evaporation bicyanide of mercury. Considering it as a compound of cyanogen and iron, it contains in 100 parts;—of cyanogen 54·4, iron 45·6.

CYANIDES OF MERCURY AND SILVER.

A full account of the poisonous properties of the cyanide of mercury (Bicyanide,) has been elsewhere given, (ante, p. 343.) From the observation of its effects on man, it appears to act more like a mercurial poison than a compound of cyanogen.

I am not aware that the cyanide of silver has ever given rise to any instance of poisoning in the human subject. It is very insoluble in water, but it is nevertheless a noxious substance. Mr. Nunneley found, in his experiments on animals, that it acted on them like hydrocyanic acid, but in a weaker degree. (Prov. Trans. N. S. iii. 86.)

Analysis.—For the analysis of cyanide of mercury, see ante, p. 343, and for that of cyanide of silver, ante, p. 547. It is only necessary to state, here, that both salts may speedily have their nature determined by the vapour-tests for prussic acid, (ante, p. 548.) Thus, half a grain (of either salt,) put into a watch-glass, and moistened with strong muriatic acid, gave the characteristic reactions with the silver and sulphur tests in a few seconds.

BITTER ALMONDS.

The bitter almond owes its poisonous properties to prussic acid, which is easily obtained from it by distillation with water. It is, however, a remarkable fact, that none of the acid exists ready formed in it, nor is the poison ever produced except by the agency of water on the almond pulp. Thus the very act of mastication produces from the pulp, the poison which destroys life: for prussic acid is formed on contact with the saliva. Heat is not required: the mere trituration of the pulp with cold water is sufficient to produce the acid.

Several cases are reported by Wibmer, in which serious symptoms occurred in children who had eaten immoderately of bitter almonds. Arzneimittel. Amygdalus.) A girl between five and six years of age, was nearly killed by eating a portion of bitter almond cake. M. Bonjean relates, that a cow was poisoned by drinking water into which a small portion of the residue left after the expression of the fixed oil had been put. (Faits Chimiques, &c. p. 56.) There are two instances recorded in which bitter almonds are reported to have caused death in the human subject, but the facts are by no means clearly detailed. Judging from reported cases, a large quantity may be taken, even by young children, without necessarily destroying life. Dr. Schlesier met with one, in which a boy between two and three years of age, ate an ounce of bitter almonds. A quarter of an hour afterwards there was a general relaxation of the limbs; the countenance was pale, depressed, and drooping; the pupils dilated; respiration sighing; there was also a tendency to sleep, followed by vomiting of the coarsely digested pulp of the almond which had a very strong smell of prussic acid. Emetics with ammonia, and exposure to a free current of air, soon restored him. (Canstatt's Jahresbericht, 1844, B. v. s. 289.)

Analysis.—The bitter almond is readily known by its shape, and the odour of its pulp. It is not easy to detect the prussic acid, evolved by the admixture of water, in the pulp of *one* bitter almond; the iron-test gives, however, a blue deposit (Prussian blue) after some hours. The pulp obtained from *one half* of a bitter almond, bruised with water, faintly affected nitrate of silver by its volatility, but in a quarter of an hour gave clear evidence of prussic acid with the hydrosulphuret of ammonia (see p. 548, ante.)

ESSENTIAL OIL OF BITTER ALMONDS. PEACH-NUT-OIL. ALMOND FLAVOUR.

The essential oil, which is produced by the distillation of the pulp of the bitter almond with water, has given rise to a great number of accidents, and has caused toxicologists to direct their attention especially to it. Its poisonous properties are entirely due to the presence of hydrocyanic acid, which is intimately combined with it. Five pounds of the almond are calculated to yield about half an ounce of the oil, and the quantity of anhydrous hydrocyanic acid contained in it, varies from eight to fourteen per cent. I find by another calculation that 2500 parts of bitter almonds yield 100 parts of amygdaline, and these by a reaction with the elements of water, produce 41 parts of essential oil and 6 parts of anhydrous prussic acid: hence 100 grains of bitter almonds would be equal to 1·88 grains of essential oil, and 0·24 grains of anhydrous prussic acid. One hundred parts of the essential oil would contain 12·76 parts of anhydrous prussic acid, and it would require 833 grains of bitter almonds to represent 100 grains of the prussic acid of the London Pharmacopœia. This oil must, therefore, be regarded as a most active poison, being at least four times as strong as the Pharmacopœial acid. One specimen which I examined was, I believe, much weaker than this average strength would indicate. Its uncertain strength renders it unfit for internal use; but in France it is given in doses of from one quarter of a drop to a drop. It becomes weaker by keeping. The oil is sold to the public in quantities of not less than a quarter of an ounce, at the rate of from four to five shillings per ounce. The liquid called Almond Flavour, spirit of almonds, or essence of peach-kernels, contains one drachm of the essential oil to one ounce of spirit. It is sold to the public in quantities of not less than a quarter of an ounce, at the rate of one shilling per ounce, for the purpose of giving a pleasant flavour to pastry! It may be as well to state that one ounce of this almond flavour is at the lowest computation equivalent in strength to 250 grains of the Pharmacopœial prussic acid. In some cases it may happen to be nearly equal in strength to this poison, and yet it is sold without restriction, and is entrusted in private families in the hands of ignorant cooks to apportion the dose which may give the requisite flavour to food!

SYMPTOMS AND APPEARANCES.—The following case by Mertzdorff will illustrate the effects produced by this poison. A hypochondriac, aged 48, swallowed two drachms of the ethereal oil of bitter almonds, and immediately threw himself on his bed. In a few minutes afterwards, having spoken to an attendant in the room and asked for water, his features became distorted, and his eyes, which were turned upwards, became fixed, and projected from their sockets. His chest heaved violently and rapidly. A medical man arrived in about twenty minutes after the poison had been swallowed, and found the patient quite unconscious, his eyes open and staring, the pupils immoveable, the respirations slow, prolonged, and accompanied with a rattling noise in the throat. The pulsations of the radial and carotid arteries, as well as of the heart itself, could scarcely be perceived, and followed each other every two seconds (30 per minute.) Swallowing was no longer possible: a strong odour of bitter almonds issued from the mouth. In ten minutes more the patient was dead, making the duration of the case *half an hour*. The body was examined twenty-nine hours after death. There was discolouration of the surface; and decomposition had advanced considerably. There flowed from the nostrils and mouth, each time the body was moved, a bloody-looking fluid, which, as well as the whole body, but especially the internal parts, emitted a powerful odour of bitter almonds: the odour was so strong as to conceal even that resulting from putrefaction. It was perceived most intensely on opening the cavity of the abdomen. In the stomach were found about six ounces of a brownish fluid which possessed the odour of bitter almonds in a marked degree. The internal surface of this organ, as well as of the small intestines, was considerably reddened. The smell of bitter almonds in the intestines, became the less obvious the further they were examined from the stomach. There was turgescence of the vessels of the brain. The blood generally was fluid, and, as well as the bile and muscles, of a dark purple or violet colour. Nothing worthy of comment in relation to the case was observed in any other part of the body. (Horn's Archiv für Mediz. Erfahr, 1823, B. ii. s. 60.) An abstract of this case will be found in the Ed. Med. and Surg. Jour. xxii. 232.

In an interesting case, the particulars of which have been communicated to me by Dr. Bull of Hereford, a woman swallowed about *seventeen drops* of the essential oil, and died in half an hour. She was seen by Dr. Bull in about fifteen minutes: her face was livid; lips separated; teeth clenched; froth about the mouth; eyes half-shut and glassy; pupils dilated and fixed; heavings of the chest at intervals; there was no pulse, and the action of the heart was scarcely perceptible. No odour was perceived about the body until after the stomach-pump had been used. On inspection nine hours after death no odour was perceptible in the cavities of the chest, head, or heart, nor in the venous blood with which the system was gorged. The organs of the chest were healthy. The vessels of the brain were congested, and there was a general effusion of serum on the hemispheres. The lining membrane of the stomach was much congested. On opening it the bitter almond odour was quite perceptible. (See Prov. Med. Jour. Sept. 11, 1844, p. 364.) The first symptoms observed in this case were strong convulsions,—the deceased throwing her arms about as if in pain. Two other cases of poisoning by this oil have recently occurred in England. A boy, aged 13, swallowed a quantity of the oil: he was found lying on the floor motionless and insensible; face pale, eyes open and fixed, pupils dilated, and he was rolling and panting for breath; the pulse at the wrist was imperceptible: the child died in a quarter of an hour without any convulsions appearing. On inspection, there was pallor of the face, with lividity of the depending parts; the lungs were congested; the odour of the poison was perceptible only in the abdomen, and very distinct in the contents of the stomach. The mucous coat of this organ was generally pale, but there were some petechial patches scattered over it. The essential oil and prussic acid were detected in it. (Lancet, July 12, 1845, 40.)

A powerful dose destroys life with very great rapidity. A man, aged 20, swallowed about two ounces of the oil. A person present saw him fall suddenly while in the act of swallowing,—he made a loud cry, gave one deep respiration, and died. A large quantity of the poison was found in the stomach, and the smell of bitter almonds was perceptible in the brain. The venous system was filled with a dark liquid blood. The lungs were healthy. (Canstatt's Jahresbericht, 1844, v. 290.) After death, even from a large dose, the odour is not always perceptible about the body. A case of poisoning by the oil of bitter almonds occurred at Hornsey, in February 1843, which may serve as an illustration, and show that an inspection is absolutely necessary in order to determine the cause of death. A chemist was found one morning lying dead on the floor of his shop. The surgeon who was first called, a few hours afterwards, suspected that the deceased had taken poison, because he saw on a shelf near the body a bottle which had contained the essential oil of bitter almonds. There was, however, no odour about the mouth, and this led to the erroneous opinion that the deceased had died from disease of the heart. The body was subsequently inspected, and it was soon rendered evident, by the powerful odour which escaped from the cavities, that the deceased had died from the effects of the oil of bitter almonds. All the viscera were in a healthy state. (Med. Gaz, April 7, 1843.)

Retention of consciousness and volition.—As in poisoning by prussic acid, a person may retain for a certain period consciousness and a power of performing certain acts. The following case, which occurred to Mr. Wakefield, is in this respect of some interest. A boy, aged 19, was sentenced by a police-magistrate to imprisonment. At twenty minutes before six P. M. he was put into the prison-van in perfect health; he reached the prison at a quarter after seven, and here, on leaving the van, he attracted the notice of the gate-keeper, who called him by his name: he answered feebly, "that's me, that's me." With slight assistance he walked to the room where the prisoners are searched, when a half-ounce bottle was found upon him labelled "Essential Oil of Almonds." It was then suspected that he had taken poison, and Mr. Wakefield was sent for. The boy stood in the room erect for five minutes. There was nothing striking or peculiar in his appearance, no smell of poison at the mouth; pupils dilated; pulse rapid and feeble; skin pallid. The stomach-pump was used, and the smell of the oil was perceptible in the liquid extracted. Ammonia was administered, and warmth applied to the extremities, but he expired just *three hours* after he had entered the van in perfect health. On inspection, the body smelt of the oil, the skin was partially livid, the blood fluid, the dura mater, as well as the lungs, gorged. The contents of the stomach had a strong smell of the essential oil of almonds, and the mucous coat, towards the pyloric orifice, had a red appearance; the other viscera were healthy. It was ascertained that the bottle had contained *two drachms* of the oil. (Lancet, Dec. 13, 1845, p. 656.) This case is remarkable in two points of view, considering the large dose swallowed,—1. the length of time which elapsed before well-marked symptoms of poisoning appeared, and—2. the length of time which the deceased survived after their appearance. In Dr. Bull's case, (ante, p. 568) there was a short interval between the taking of the poison and the production of insensibility. The deceased called out, and she had had time to cork a small bottle which had contained the poison, to put it into a bag, draw the strings of the bag, and hang it over a chair by the side of the bed.

In October, 1845, a case was referred to me for examination by Mr. Savage, in which there was also clear evidence of the power of locomotion after probably a large dose of this poison. The deceased mixed the poison with some ale, in a cup, stirred it up with a pipe, and drank off the greater part. Five minutes had elapsed when he was seen deliberately walking towards a staircase conscious and self-possessed, for he replied rationally to questions put to him. The symptoms then appear to have come on very suddenly, and to have commenced with vomiting, during which, probably, part of the oil which he had swallowed, was ejected. He became insensible, and the breathing, as usual, was convulsive and took place at intervals; but, excepting slight opisthotonos, there were no convulsions. From the facts observed by Mr. Savage, it appears probable that the whole duration of the case did not exceed *seven minutes;* and the fatal symptoms were not manifested until within the last *two minutes.* On a post-mortem inspection there was pallor of the face: cadaverous rigidity (eighteen hours after death) was strongly developed: and the hands and feet were unusually livid. The eyes were remarkably brilliant. The blood, with which the venous system was gorged, was liquid and of a dark colour. The lungs, as well as the heart, were quite healthy. There was an odour of the oil of bitter almonds on opening the chest, rendering it probable that either a portion of the essential oil, or its odoriferous principle, had been absorbed with the prussic acid and circulated throughout the body. The brain, which was perfectly healthy and free from congestion, gave out a slight odour of bitter almonds. This was also perceived in the cavity of the chest. The mucous membrane of the stomach was reddened to a degree which may be regarded as somewhat unusual. Mr. Savage described it as much inflamed about the cardiac extremity and œsophageal opening. When I saw it, (ten days after death) although the stomach had probably undergone some change, the cardiac extremity of the organ appeared as if it had been acted upon by a powerful irritant. It is possible that the redness may have depended on previous disease; but the essential oil of bitter almonds has a hot and burning taste, even when freed from prussic acid; and, like some other essential oils, it may exert an irritant action when taken in a large dose. Prussic acid was detected in the contents of the stomach, and the odour of bitter almonds was very powerful in this organ so late as *ten days* after death.

QUANTITY REQUIRED TO DESTROY LIFE.—In determining this question it is necessary to bear in mind that the essential oil varies much in strength. Probably in no case is it of weaker strength than Scheele's prussic acid (ante, p. 518,) while in general it will be found to be from four to six or eight times the strength of the acid of the London Pharmacopœia. Sir B. Brodie, with the design of tasting it, applied *one drop* to his tongue. He immediately felt a remarkable and unpleasant sensation in the epigastrium, with such weakness in the limbs and loss of power in the muscles that he thought he should have fallen. (Paris, Med. Jur. ii. 404.) This proves that it is dangerous in the smallest doses, and quite unfit for medicinal use. The smallest quantity of the oil which has yet been known to destroy life was in the case which occurred to Dr. Bull of Hereford. A woman, aged 49, was in this instance killed in half an hour by a dose of less than twenty drops. Probably not more than *seventeen drops* were taken. (See ante, p. 568.) Dr. Bull informed me that from the result of his minute inquiries, he is satisfied that the dose did not exceed this quantity. An adult has recovered, although with some difficulty, from a dose equivalent to *thirty drops* of the essential oil. Mr. P. H. Chavasse of Birmingham, has reported this case, which is in other respects of great interest. A druggist swallowed half an ounce of almond flavour (equivalent to thirty drops of oil.) Mr. Chavassè saw him ten minutes afterwards. His symptoms were very peculiar. In less than half a minute after he had swallowed the poison, he fell down in a state of syncope, his face being deadly pale, and his pulse (according to a by-stander) quite imperceptible. After a few minutes he came to himself, and he was then put to bed. The moment he recovered from the fainting, he vomited some undigested food and bile, *strongly impregnated with the odour of prussic acid.* He then became delirious muttering to himself, and speaking almost incoherently. In a very short time the delirium ceased; the whole frame then became slightly convulsed, especially the upper eyelids. The convulsions in a minute or two ceased, with the exception of the eyelids; these continued convulsed nearly the whole time Mr. Chavasse was with him. For a few minutes *he was sensible,* and spoke on the nature of his attack, but again gradually relapsed into a delirious state; his whole face was lit up with an expression of excessive joy; his eyes shone brilliantly; indeed, he exhibited the appearance of one who had been inhaling the laughing gas. These symptoms continued for a few minutes and he again became sensible, and expressed himself as being much better. The pulse which was before quick and intermittent, now became more slow and regular; the expression of the face assumed a more natural aspect, with the exception of the eyes, which throughout the attack continued extremely brilliant. While the attack lasted the respiration was excessively short, and when the patient was rational he was fearful every moment that he should be suffocated. The body during the whole time was cold. He gradually recovered from the effects of the poison. (This case is reported in the Lancet, Sept. 1839, p. 930. An erroneous version of it appeared in the Journal de Chimie Médicale, representing the reporter as M. Chavasse, and the dose swallowed as *half an ounce* of the oil!) These two cases, I believe, comprise the largest dose from which a person has escaped, and the smallest dose which has yet been known to prove fatal.

Mr. Chavasse's case is especially remarkable in the fact that, so far as I can ascertain, it is the only instance of a temporary *remission* of symptoms in this form of poisoning by prussic acid. Considering the fatal effects produced by a dose of less than twenty drops, and the severity of the symptoms, from which the person only recovered under active and immediate treatment, after taking thirty drops, we may infer that a dose of from *twenty* to *forty drops* of the oil may prove fatal to adults under common circumstances. Children would die from a still smaller quantity; nevertheless, an interesting case is reported, in which a child between eight and nine years of age recovered from a dose equivalent to seven drops. A girl swallowed about a teaspoonful of a mixture sold by druggists as "ratafia," composed of one part of the essential oil of bitter almonds to seven parts of spirit. The quantity swallowed by the patient was equivalent to about *seven drops* of the essential oil. When seen immediately after the accident, there was complete insensibiliiy; the eyelids were closed, but the eyes were brilliant and glassy, without any mental expression; the pupils dilated, no pulse at the wrist; the carotids beating fully and quickly; relaxation of the muscles of the extremities, but the lower jaw was clenched in rigid spasm. Cold affusion with stimulants, stimulating frictions and emetics, were employed. Vomiting was induced, and the ejecta had a strong smell of prussic acid. In about twenty minutes the pulse returned,—the child opened her eyes, and was able to answer questions. This case shows that a small dose of the oil may give rise to very alarming symptoms; and it is probable, that but for the active and prompt treatment adopted, the child would have died. (Lancet, June 1844.)

A case occurred at Guy's Hospital in May 1843, in which a boy, aged 12, recovered under immediate treatment. The boy was accosted in the street by another boy, who had a medicine basket on his arm, and from this he took a bottle and offered to the patient some liquid which he called almond oil. He thought that he swallowed about a table-spoonful; he experienced shortly afterwards a burning sensation in his throat, and in about ten minutes, he sat down on a door-step, and became insensible. In about eight minutes afterwards he was brought to the hospital. His breath smelt strongly of bitter almonds; there were violent tetanic convulsions, with complete opisthotonos; the head and neck being drawn backwards, the elbows drawn behind his back, and firmly fixed in this position. The jaws were quite fixed; there was complete insensibility, and the pulse was scarcely perceptible. The treatment consisted in cold affusion to the spine and the use of the stomach-pump. The stomach was well washed out with a large quantity of water, and this smelt strongly of bitter almonds. During the treatment, the patient suffered from strong convulsive twitchings of the muscles. In about an hour he recovered, and in the course of a few hours left the hospital. It is very probable that in this case the boy swallowed a portion of what is called almond flavour, a diluted solution of the essential oil. The contents of the stomach were submitted to two distillations, and about four ounces of a clear liquid, smelling strongly of bitter almonds, were procured. This liquid was scarcely rendered cloudy by nitrate of silver, and the iron test gave no trace of Prussian blue. The only proof, therefore, of the nature of the poison, was, the odour of the essential oil, which was very powerful, notwithstanding the want of action in the tests.

Local action.—Like prussic acid, the essential oil of bitter almonds may have a local action. In small quantities it would act as a sedative; but, from its greater strength, its operation would be, *cæteris paribus*, more violent than that of prussic acid. It is proper to mention, that this oil is employed in the preparation of numerous cosmetics which are applied to the skin, and no attempt is made to separate the active poison from it before it is thus used! These cosmetics, which are extensively sold by perfumers, are to be regarded as highly poisonous compounds, the use of which for external application should be strictly prohibited. Local paralysis might easily arise from their employment.

Is the vapour of the oil sufficient to produce fatal effects?—This question was raised in the subjoined case, which occurred in London, in 1838. The deceased, the wife of a publican, had been clearing out a closet, which contained, among other liquids, a bottle of the essential oil of bitter almonds. She was suddenly heard to call out. A servant found her pale and faint, and she complained of sickness. There was a strong odour in the room, and deceased said that the corks of some of the bottles had come out, and the smell had made her feel sick. She was removed to bed, but died before any medical assistance could be obtained. There was no motive for the deceased committing suicide, and it was a subject of inquiry, whether the vapour alone might not have caused death. This question was set at rest by an inspection of the body; some of the poison was found in the stomach, and there was a very strong odour of bitter almonds in the contents. It was therefore clear that the deceased must have swallowed a portion of the oil,—whether from motives of curiosity or not, it is impossible to say. The vapour may produce vertigo and stupor; but unless long respired, it would not be likely to cause fatal effects.

Chemical analysis.—This oil, which is often called peach-nut oil, is colourless when pure, but it commonly has a pale yellow colour, and a strong odour of bitter almonds, by which it is at once identified. It has a hot and burning taste. It gives a greasy stain when dropped on paper, which does not entirely disappear on the application of heat. It sinks in water, and readily combines with alcohol and ether. If to the alcoholic liquid a small quantity of caustic potash, and a solution of green sulphate of iron be added, Prussian blue is formed on agitating the mixture, but does not appear until the precipitated oxide of iron is dissolved by the addition of diluted sulphuric or muriatic acid. One drop of the oil is sufficient for this experiment. Water will separate a small portion of prussic acid from the oil. Thus, by agitating in a tube about one drachm of the oil, with three or four drachms of distilled water, and after a few minutes, filtering through a *wet* filter, the oil is entirely separated. The liquid which passes through is scarcely acid; it is rendered cloudy by nitrate of silver, and gives a decided blue-coloured precipitate with the sulphate of iron and caustic potash; it possesses all the properties of a weak solution of prussic acid. The oil and prussic acid may be more completely separated by distillation with lime and water, although it is probable that all the prussic acid is not obtained by this process. Nevertheless, if the aqueous product of the distillation be filtered through a wet filter, a clear liquid is obtained, giving an abundant precipitate with the silver test, and well-marked effects with the sulphate of iron and potash. It is worthy of remark, that the filtered liquid, after entire precipitation by nitrate of silver and oxide of iron, has the odour of bitter almonds just as strongly as before; and that the water holds some organic matter, is proved by the surplus nitrate of silver causing it to assume a violet tint when it is exposed to the light of the sun. This odour is so persistent, that it will commonly be found in the body for some days after death. In addition to the processes above mentioned, the vapour-tests speedily indicate the presence of prussic acid, especially the hydrosulphuret of ammonia in *Organic liquids.* The oil, owing to its density, is generally found at the bottom of the vessel; it may be separated by decantation, distillation, or by agitating the liquid with its bulk of ether, and then pouring off the ethereal solution.

The essential oil, deprived of prussic acid, and perfectly pure, does not act injuriously. (Pharm. Jour., July 1847, 12.)

BITTER-ALMOND WATER.

This water is made by distilling one part of the almond-cake with eight parts of water. It varies considerably in strength. Gregory states that it contains one per cent. of anhydrous prussic acid. Mr. Bell informs me that in a specimen which he analyzed, the proportion of acid was 0·27 per cent. I have met with specimens containing less than this. The odour is no criterion of the strength, since the odour of prussic acid is concealed by that of the bitter almond. Its strength is impaired by keeping; thus Zeller found that one ounce of the water fresh made, yielded 5·12 grains of cyanide of silver; but after one year, when merely corked in a bottle, the proportion yielded was only 4·62 grains. (Pharmaceutical Journal, February 1846, 371.) The liquid is poisonous, and a trial recently occurred in this metropolis (*Reg.* v. *Cronin*, C. C. C., April 1847,) which attracted much attention to the subject. The accused was charged with the manslaughter of a female under the following circumstances. He had been in the habit of using a preparation which he called bitter-almond water, made by mixing three drops of the essential oil with a pint of water—a harmless mixture in small doses. He wrote a prescription for the deceased, in which occurred the words *aquæ amygd. amar.* Six ounces of this were ordered, and the mixture contained besides a very small dose of prussic acid. The chemist who prepared the mixture put into it six ounces of the liquid commonly known as bitter-almond water (distilled from the cake.) The deceased took a tablespoonful and a half. In three minutes she said, "Oh, how queer I feel!" She left the room, and ran out towards the garden, where she fell, breathing hard and groaning. There was dilatation of the pupils, with general relaxation of the limbs, but there were no convulsions. She died shortly afterwards. There was no doubt that the bitter-almond water had caused her death. The viscera were generally healthy. There was no odour in the abdomen, but it was perceptible in the brain,—the vessels of which were somewhat congested. Prussic acid was detected in the stomach. The accused was acquitted, as it was not considered that he was strictly responsible for the result. (Med. Gaz. xxxix. 388 and 695.) The quantity of prussic acid which the deceased took was equivalent to 0·94 of the anhydrous compound,—thus bearing out in a most striking degree, the assumed fatal dose (ante, p. 539,) and proving that dilution does not prevent the rapid action of this poison (p. 526, ante.)

This water is not commonly employed medicinally in England, as its effects are very uncertain. Eighteen drops have been known to produce vertigo, dimness of sight, and tendency to sleep. Twenty-two drops produced convulsions and vomiting. MM. Duvignan and Parent, who tried these experiments on themselves, did not feel inclined to carry the dose further. A drachm of the water killed a moderate-sized dog. (Paris, Med. Jur., ii. 403.) In France bitter-almond water is used medicinally, in doses of from ten to forty drops.

Analysis.—The water is sometimes opaque, from a little oily matter diffused in it, but it may be rendered clear by alcohol. Some specimens will give readily all the usual reactions with the liquid tests for prussic acid; but when the water has been long kept, it has often only the odour of the bitter almond, and none of the acid. The uncertainty of its composition will be rendered evident by the following results. Two specimens of the water, prepared by the distillation of bruised bitter almonds with water, were tried merely by volatility; as it was certain that if this experiment succeeded, the mixture of liquids could not possibly fail. With one specimen, after half an hour, no effect had been produced on nitrate of silver; and on trying the hydrosulphuret of ammonia no sulphocyanate of ammonia was detected in the evaporated residue. Two drachms of the water gave no perceptible precipitate with nitrate of silver, nor could any Prussian blue be obtained by adding the iron-test to a similar quantity of liquid. The second specimen of bitter-almond water, which was slightly precipitated by nitrate of silver, and affected that liquid by its volatility, gave decided evidence of prussic acid in *three minutes* by the sulphur-test for prussic acid (ante, p. 548.) The strength of the bitter-almond water is by no means proportioned to the quantity of the bitter-almond used, but it varies according to the process employed for its production. The same weight of almonds has given two kinds of water,—one ounce of one giving as much as 5·35 grains of cyanide of silver, and one ounce of the other only 2·5. When the residuary almond-cake has been previously digested in spirit, the water obtained is always weaker, (Pharm. Journ., Feb. 1846, 371.)

LAUREL OIL. LAUREL WATER. CHERRY LAUREL WATER.

This is a very weak solution of prussic acid, containing only about one-fourth of a grain per cent. of the strong acid; but it is said to be more poisonous than this quantity of acid would indicate. (Pereira, ii. 1542.) The leaves gathered in wet and cold weather are found to yield more hydrocyanic acid than those gathered in hot and dry weather. (Zeller.) In some specimens, which I procured by distilling the bruised tops and fine shoots of the laurel with water, the odour was powerful, but the proportion of prussic acid present was considerably less than that above stated. Like bitter-almond water, it is probably very variable in strength. Specimens long kept and frequently exposed seldom contain any prussic acid, although the odour of bitter almonds is strong. It is a limpid colourless liquid, producing, in large doses, the usual effects of poisoning by prussic acid.

By distillation with water, the leaves of the plant yield also an essential oil, CHERRY LAUREL OIL, resembling that of the bitter almond, but much weaker, as it contains on an average less than three per cent. of prussic acid. According to Christison, almost every part of the plant is poisonous, but especially the leaves, flowers, and kernels; but the pulp of the cherry is not poisonous. Articles of food are often flavoured with the leaves, and accidents are said to have arisen from this practice. (Pharm. Journ. July 1847, 13.) Dr. Paris states that several children were severely affected by partaking of some custard flavoured with the leaves of the laurel, and were ill for three days. A girl of six and a boy of five years of age fell into a profound sleep, out of which they could not be roused for ten hours. (Med. Jour. ii. 402.) It is often employed with impunity; but then it appears that the proportion of oil and prussic acid is liable to vary with the age of the leaf. Dr. Christison states that he has found ten times as much oil in the young as in the old leaves, when both were gathered in May and June, (Op. cit. 788.)

Cases of poisoning by laurel water are not common, and generally arise from accident. In a former part of this work, I have referred to the memorable trial of Captain Donellan, in 1781, on a charge of poisoning Sir T. Boughton, by this liquid. This trial is of great interest to the medical jurist. The prisoner, it is supposed, substituted for a purgative draught, two ounces of laurel water. Admitting that the laurel water had no greater strength than that just now assigned to it, the deceased must have taken 2·4 grains of pure hydrocyanic acid, a quantity equal to *fifty drops* of Scheele's prussic acid. The draught was administered to the deceased by his mother, Lady Boughton; she perceived that it smelt strongly of *bitter almonds*—the only evidence of the probable nature of the poison; for the original draught, containing rhubarb, jalap, spirits of lavender, and nutmeg water, would have had no such smell. The following were the symptoms: "In about two minutes after swallowing the draught, the deceased appeared to struggle very much, as if to keep it down, and had 'a rattling and gurgling' at his stomach. In about ten minutes, he seemed inclined to doze, and in about five minutes afterwards, he was found with his eyes fixed upwards, his teeth clenched, and froth running out of his mouth." He died in half an hour after swallowing the draught. The post-mortem examination proved nothing (ante, p. 102;) no poison was detected in the body, but the inspection was not made until eleven days after death.

In making every allowance for such coincidences, in the supervention of fatal disease at the time of taking medicine or food, as have elsewhere been pointed out, I do not think there is any reason to doubt that in this case the deceased was poisoned and the prisoner properly convicted. It has been urged that the medical evidence was of itself insufficient; and that without the moral circumstances, the charge of poisoning could not have been made out. But it is impossible to divide evidence in this way; it is like separating two series of circumstances in presumptive evidence, either of which taken singly may be weak, but when taken together, become strong. Many convictions on medico-legal trials for murder by poisoning, would not have occurred if the fact had rested on moral or on medical evidence *alone*. In Donellan's case, the medical evidence was strong, whether we regard the time of the occurrence of symptoms, their character, or the period within which death took place. To exclude all notion of these effects depending on a draught just before taken, and having the decided odour of a liquid known to be capable of producing them; an odour which the originally prescribed draught could not possibly have had,—and to refer them to a disease, unusual in so young a subject, and unlikely to have caused death so rapidly, or under the symptoms witnessed, is to create impunity for the cunning and skill often displayed in murder by poison. *Direct* evidence can rarely be obtained in such cases,—the murderer, unless insane, does not proclaim to the world his intention to poison another, nor the nature of the poison used, nor does he administer it openly. Every minute circumstance, therefore, requires the closest watching and analysis, if we wish to prevent by punishment, this most detestable crime. In several cases, which have occurred since that of Donellan, the medical evidence of poisoning has not been stronger; but taken, as it always ought to be, with moral circumstances, it has been held sufficiently strong by Courts of Law for a conviction of the accused party.

The following is a recent instance of poisoning by laurel water. About half a teaspoonful of a mixture, consisting of four-fifths cherry-laurel water, was given by mistake to an infant eight months old. The child threw its head back, was convulsed, and died in a few seconds. The laurel water taken in this case, is said to have been stronger than usual. The body was inspected twenty-four hours after death. Nothing was observed in the brain and spinal marrow, but the stomach contained two teaspoonfuls of a yellowish liquid without odour; and its mucous membrane was injected towards the greater curvature. No trace of prussic acid was found in the contents, but the poison was easily detected in the liquid remaining in the phial. (Med. Gaz. Jan. 1843.)

The next case is remarkable chiefly from the circumstance of the symptoms coming on very slowly. A man swallowed one morning an ounce and a half of laurel water. No symptoms appeared until three hours afterwards. There was then a numbness of the hands and feet, drooping of the head, and involuntary expulsion of the urine and fæces. The extremities became cold, and he lost all power over them, although the sensibility was retained. The pulse was small: there was perfect consciousness. He gradually became weaker, and died the same evening. On inspection the only remarkable appearance was—that the blood was viscid and of a dark colour. There was no odour of bitter almonds. (Canstatt, Jahresbericht, 1844, v. 289.) This is an anomalous case: it is quite unlike the effects produced by laurel water.

In death from over-doses of this water, the symptoms and appearances are precisely the same as in poisoning by diluted prussic acid. The same treatment is required (ante, page 540.)

ANALYSIS.—The odour of the water is sufficient to identify it, but this will not prove that it contains prussic acid. In order to obtain this proof, it must be submitted to analysis. The following is the result of the examination of a very weak specimen:—Nitrate of silver produced no perceptible effect with one drachm of it when the liquids were mixed, nor could any Prussian blue be procured from a like quantity by the use of the iron-test. One drop of hydrosulphuret of ammonia, added to three drops of the water, gave the clearest evidence of prussic acid by the production of the red sulphocyanate of iron, when the persulphate was added to the evaporated residue. From five to ten drops placed in a watch-glass, produced no film (by the vapour) on nitrate of silver after the lapse of twenty minutes: in the same period of time, one drop of hydrosulphuret of ammonia absorbed the vapour, and left, on evaporation, a perceptible quantity of sulphocyanate. Prussic acid is also easily detected by the sulphur-test in the bruised shoot of the laurel. The strength of this water is so variable, that it admits of no safe comparison with prussic acid: each specimen requires to be separately examined. An *aqua lauro-cerasi* is used in the Dublin and Edinburgh Pharmacopœias. The French codex prescribes the dose of from ten to forty drops every two hours. (Pharm. Jour. Feb. 1846, 372.)

The distilled water of the leaves of the *Acacia* contains prussic acid. The water has a strong smell of bitter almonds, and eight ounces of it, precipitated by nitrate of silver, yielded 4·15 grains of cyanide. The dried leaves gave no prussic acid on distillation. The leaves of the peach yield a water as strong as that of the laurel. The distilled water of the *leaves* of the *Sweet almond* contains prussic acid. Zeller found that one ounce gave 0·575 grains of cyanide of silver. The flower of the common lilac is also said to yield traces of this poison.

NOYAU, CHERRY RATAFIA.

These, and all other liqueurs having the smell of bitter almonds, are considered to be poisonous when taken in large doses. The quantity of prussic acid present in them is liable to vary; it may be separated by distillation at a gentle heat, and then tested. I have found that an ounce and a half of good noyau, having a strong odour and flavour, yielded when distilled to two-thirds, scarcely a trace of prussic acid either by the silver or iron test. It had been kept some years in a well-corked bottle. An equal quantity of cherry ratafia, similarly treated, gave me no ponderable quantity of Prussian blue.

There are other plants, the leaves and kernels of which yield prussic acid; these are, the Bird-cherry, the Peach, Nectarine, Damson, Mountain-ash, Apricot, and the seeds of apples and pears. I have examined the seeds of oranges and figs, but have found none; nor could I obtain the slightest trace of prussic acid from the distillation of three hundred grains of the *sweet* almond. The quantity produced from the seeds of apples has been much exaggerated. I found that the seeds of ten common apples distilled with water, with their skins *unbroken*, yielded not a trace of prussic acid. When reduced to a fine pulp and distilled with a small quantity of water,—nitrate of silver scarcely rendered the liquid cloudy, and the quantity of Prussian blue obtained from the whole, was so small that it was difficult to assign an estimate to its weight. Taking it at the maximum it could not have exceeded the 150th part of a grain! The experiment was repeated with a like result. I should much doubt, from my experiments, whether the seeds of twenty apples would yield so much prussic acid as one bitter almond. The apples for these experiments were procured indiscriminately from a public market. It is possible that the proportion of prussic acid obtained may vary in different apples, but it must be in all cases small; and in no case is it likely to be formed unless the seeds are buised or well masticated. (See Med. Gaz. xxxvi. p. 328.) It is also a fair question, whether, unless the seeds were picked out and eaten separately, a person would not die from the mechanical effects of the apples before the prussic acid could be evolved in sufficient quantity to do injury to him!

CHERRY AND PEACH KERNELS.

Fresh and dried cherries, as well as the kernels and stones, yield prussic acid by distillation. The quantity yielded by the pulp of the cherry is exceedingly small, amounting to mere traces, but it is much greater in the stones and kernels. From sixteen ounces of cherry-stone water, Geiseler obtained 1·9 grains of cyanide of silver; and from cherry-kernel water, the kernels being to the water as 1 : 8 by weight, the cyanide of silver obtained from sixteen ounces, was equal to 2·36 grains. Twelve ounces of the *kernels* yielded 7· grains of hydrocyanic acid: but the proportion of prussic acid yielded by the same weight of cherry *stones*, according to Geiseler, was not more than 2·3 grains. (Pharm. Jour. Feb. 1846, 372.) These kernels bruised are much employed for the purpose of giving a flavour to alcoholic [illegible]. It is not often that they are used in such quantity as to [illegible] symptoms: but the following case, the details of which are somewhat imperfectly given, will show that the eating of a large quantity of the kernels may operate fatally.

A girl aged five years ate a considerable quantity of the kernels of black cherries (prunus avium). Her brother (a few years older than herself), also ate some. After the lapse of a few hours, symptoms of poisoning appeared. When a medical man was called the next day, he found the girl in such a stupor, that she could not be roused. The eyes were closed, pupils moderately dilated, the skin moist and hot, respiration exceedingly hurried, pulse small and quick, urine and fæces discharged involuntarily, the child very restless. An effervescing mixture was ordered internally, and cold fomentations to the head externally; after a few hours, vomiting of a greenish mass ensued, and was followed by retching, which continued until death: the body was spasmodically drawn backwards. The [illegible] lived [illegible] hours. On a *post-mortem* examination, the stomach was found intensely inflamed: the intestines were strictured and invaginated, but there was not any inflammation. The liver, spleen, and large vessels, contained black tar-like blood. The boy who had eaten fewer cherry-kernels, became likewise ill, but recovered in the course of a month. An eruption, analogous to urticaria, came out on the fore-arms of both children; they were both perfectly well (according to the statement of the mother) before eating the cherry-kernels, and no other cause for the attack could be assigned. The kernel of the prunus avium (cerasus nigra) contains amygdaline, and produces prussic acid as well as essential oil in the stomach. (Philadelphia Med. Exam. July 1845. [illegible].)

[Accidents have occurred from eating the fruit of the wild cherry. *Cerasus serotinus and C. Virginianus.*) According to the experiments of Mr. Procter (*Am. Jour. Pharm.* iii. 191,) Prussic acid exists in the kernels of these fruits in some quantity.—G.]

A singular case of poisoning by *peach-kernels* has been communicated to me by Mr. Hicks of Newington. A medical man swallowed half an ounce of liquid made by digesting gin on a large quantity of peach-kernels. He became giddy and had violent constriction of the fauces and dimness of sight. He vomited and recovered. The bottle was brought to me by Mr. Hicks.—a few drops of the liquid contained in it, yielded only a faint trace of prussian blue. The kernels weighed 124 grains; they were large and the skins entire. All the prussic acid must have been extracted, either as such or under the form of amygdaline; for on bruising the kernels and distilling them with water not a particle of the poison could be procured!

JATROPHA MANIHOT.

The root of one variety of this West Indian plant, known under the name of Bitter Cassava contains in its juice, prussic acid. It is, therefore, when recently expressed, highly poisonous, inducing coma, convulsions, and death. Prussian blue is easily obtained from the fresh juice by the iron-test for prussic acid. The vegetable principles of the plant, evaporated to dryness, form what is called *Cassava-cake*, which is not only inert, by reason of the poison being volatilized, but highly nutritious. The starch obtained from this root is well known under the name of *Tapioca*. Neither cassava nor tapioca yields any trace of prussic acid.

CHAPTER XL.

NARCOTIC POISONS CONTINUED.—HYOSCYAMUS NIGER. SYMPTOMS AND EFFECTS—CASES—LACTUCA VIROSA AND SATIVA—LETTUCE-OPIUM—SOLANUM DULCAMARA AND NIGRUM—SOLANINE—CAMPHOR—SYMPTOMS AND APPEARANCES—CASES—ANALYSIS—ALCOHOL—SYMPTOMS—EFFECT OF DILUTION—POISONING BY THE VAPOUR—DIAGNOSIS—FATAL CASES—TREATMENT—ANALYSIS—ABSORPTION—POISONING OF ALCOHOLIC LIQUIDS—ACTION OF ETHER AS A LIQUID—SYMPTOMS AND APPEARANCES—EFFECTS OF ETHER-VAPOUR—CASES OF POISONING BY—CHEMICAL ANALYSIS—ABSORPTION AND ELIMINATION—ITS ACTION ON THE BLOOD.

There are various other poisons which, from their acting especially upon the brain and nervous system, require consideration under this division of the work. They are very rarely used for the purpose of destroying life, but, as the result of accident, they sometimes give rise to fatal effects.

HYOSCYAMUS NIGER.

All the parts of this plant, which is commonly known under the name of HENBANE, are poisonous. The seeds produce the most powerful effects, then the roots, and lastly, the leaves. The vapour evolved from the fresh-cut leaves, has been known to produce vertigo, stupor, and syncope. In small or medicinal doses, henbane has a narcotic action; but when taken in large doses, it produces those effects usually assigned to the narcotico-irritant class.

Symptoms and appearances.—The best summary of these is given by Wibmer, (Arzneimittel, Art. HYOSCYAMUS NIGER.) When the dose is not sufficient to destroy life, the symptoms are,—general excitement, fulness of the pulse, flushing of the face, weight in the head, giddiness, loss of power and tremulous motion of the limbs, somnolency, dilatation of the pupils, double vision, nausea and vomiting. After a time these symptoms pass off, leaving the individual merely languid. When a large quantity of the root or leaves has been eaten, an accident which has occurred from the plant having been mistaken for other vegetables, then other and more serious effects are manifested. In addition to the above symptoms in an aggravated form, there will be loss or incoherency of speech, delirium, confusion of thought, insensibility, coma, and, sometimes, a state resembling insanity; the pupils are dilated, and insensible to light, there is coldness of the surface, cold perspiration, loss of power in the legs, alternating with tetanic rigidity and convulsive movements of the muscles, the pulse small, frequent, and irregular, the respiration deep and laborious. Occasionally there is nausea, with vomiting and diarrhœa. Death takes place in a few hours or days, according to the severity of the symptoms. The special effect of this poisonous plant is manifested in its tendency to produce a general paralysis of the nervous system. As an instance of the singular train of symptoms occasionally produced by it, Dr. Houlton states, that in a monastery where the roots had been eaten for supper by mistake, the monks who partook of them were seized in the night with the most extraordinary hallucinations, so that the place became like a lunatic asylum. One monk rang the bell for matins at twelve o'clock at night: of those of the fraternity who attended to the summons, some could not read, some read what was not in the book, and some saw the letters running about the page like so many ants! (Lancet, July 6, 1844, 479.)

When the extract or decoction is introduced into the rectum, or applied externally to a wound, similar effects are observed to follow.

After death there is a general congestion of blood in the venous system, especially in the brain: the lungs are gorged. There are commonly no marks of irritation in the alimentary canal.

Among the reported cases of poisoning by hyoscyamus is the following. A woman collected in a field, a quantity of the root by mistake for parsnips. They were boiled in soup, of which nine persons in the family partook without remarking any particular taste. Very shortly afterwards, the whole of these persons felt uneasy, and complained of a bitter acrid taste in the mouth, with nausea. The pupils of the eyes were dilated, and there was indistinctness of vision. These symptoms were followed by great restlessness, convulsions, and continued delirium. The patients successively lost the power of vision, hearing and voice, and were affected with stupor and insurmountable somnolency. (Ed. Med. and S. J. Oct. 1844, 562.)

Orfila relates the cases of two men who ate the young shoots of the plant. The first effect was, that the earth seemed to pass from under them: the tongue became paralyzed, and their limbs cold, torpid, paralysed, and insensible; the arms in a state of spasmodic action, the pupils were dilated, the look was fixed and vacant, respiration difficult, the pulse small and intermittent. Besides these symptoms, there was *risus sardonicus*, with delirium; and the jaws were spasmodically closed. Under treatment, the men recovered in the course of two days. (Op. cit. ii. 264.) A decoction of the plant, introduced as an enema, produced somewhat similar symptoms, although in a less severe form. The patient appeared in this instance as if attacked with apoplexy, except that there was no stertorous breathing.

There are no data by which we can determine the relative activity of Hyoscyamus. In powder the medicinal dose of the leaves is from five to ten grains; of the seeds, from three to eight grains. The dose of the tincture is from half a drachm to two drachms, and of the extract, from five to ten grains; but this preparation is more likely to vary in strength than any of the others. Dr. Burder states, that he has observed great inconvenience to follow from a dose of ten minims of the tincture repeated every six hours. After three or four doses there was pain, with oppression of the head. Ten minims given in equal doses at an interval of six hours, were followed by pain of the head, flashing of light before the eyes, and delirium. (Lancet, July 6, 1844, 480.) There may be, as in the case of opium, an idiosyncrasy with respect to this drug.

One fatal case of poisoning by the roots is quoted by Orfila, and another by the leaves, by Wibmer. Twenty seeds have produced complete delirium, (Wibmer, Op. cit. 147,) and the same writer states, that, in one instance, very alarming symptoms were caused by seven grains of the extract (154.) The poisonous properties of the plant are affected by soil and season. They are most developed in it, while the seeds are being formed.

Treatment.—The speedy expulsion of the poison by emetics.

Analysis.—The poisonous properties of Hyoscyamus are known to be owing to a crystalline alkaloidal body, which is called Hyoscyamia. It is very difficult of extraction. The crystals have a silky lustre,—they are not very soluble in water, but are easily dissolved by alcohol and ether. It has an alkaline reaction, and its solution is precipitated by tannin. It has an acrid disagreeable taste, resembling that of tobacco. It is highly poisonous, and causes dilatation of the pupils. When the vegetable has been eaten, it can be identified only by its botanical characters. The seeds are very small and hard, they are honey-combed on the surface, and may easily be confounded with those of belladonna.

There are other varieties of this plant which are also poisonous.

LACTUCA.

The two species of lettuce, known under the names of LACTUCA SATIVA and VIROSA, contain a principle which is possessed of feebly narcotic properties. Orfila has found that the extract prepared by evaporation at a low temperature, acts upon the brain and nervous system of animals; although very large doses were required for the production of narcotic effects. There is no record of these plants having exerted a poisonous action in the human subject.

Analysis.—The inspissated juice of the lettuce, is well known under the name of LACTUCARIUM or LETTUCE-OPIUM. The Lactuca Virosa yields three times as much as the Lactuca Sativa; and half a grain of it, according to Dr. Fisher, is equivalent to two or three grains of that obtained from the Lactuca Sativa. (Med. Gaz. xxv. 862.) The juice, when it first escapes, is of a milky-white hue, but, in drying, it forms an extract in small irregular dry masses of a brown colour, a bitter taste, and with an odour similar to that of opium. It has a weak narcotic action when given in doses of from five to twenty grains. It varies much in strength. Wibmer found that *two grains* caused headach and somnolency. (Op. cit. 200.) By the smell only, it is liable to be mistaken for opium. It is but little soluble in water, and after long boiling, it forms a brown turbid solution which gives a greenish tint with sesquichloride of iron. It therefore contains no meconic acid. On examining a good specimen I have not found any trace of morphia. This shows that the odour of opium may exist in substances which do not contain meconate of morphia. Nitric acid gives a yellowish tinge to the decoction, as it does to most other vegetable solutions. It is bitter to the taste, which appears to be owing to the presence of a bitter principle called LACTUCIN, upon which its feebly narcotic properties probably depend. There are no tests for lactucarium, further than the colour, the opiate odour with the want of solubility, and the absence of the other chemical characters of opium. In the plant, it is combined with malic acid, potash, and resin. (Fisher, loc. cit.)

SOLANUM.

There are two species of this plant—the Solanum Dulcamara, *Bitter-sweet* or *Woody-nightshade*, which has a purple flower and bears red berries; and the Solanum Nigrum, or *Garden-nightshade*, with a white flower and black berries. Dunal gave to dogs four ounces of the aqueous extract, and, in another experiment, 180 ripe berries of the Dulcamara, without any ill effects resulting. On the other hand, Floyer states that thirty of the berries killed a dog in three hours. (Wibmer, op. cit. SOLANUM.) These differences may perhaps be reconciled by supposing that the active principle *Solania*, on which the poisonous properties of both species depend, varies in proportion at different seasons of the year. In one instance a decoction of the plant is said to have produced in a man dimness of sight, vertigo, and trembling of the limbs,—symptoms which soon disappeared under slight treatment. [In some parts of our country the Celastrus Scandens is known under the name of Bitter-sweet, and its berries are apparently more active than those of the S. Dulcamara.—G.] Orfila found that the extract of Solanum nigrum had a very feeble effect as a poison: and the fatal cases reported to have been caused by it, are perhaps properly referable to belladonna, for which it may have been mistaken. The single death from Dulcamara reported in the Registration returns for 1840, may have been due to a mistake of this kind.

Nevertheless the berries of the Solanum nigrum, in one instance, at least, produced very serious effects in three children who had eaten them. They complained of headach, vertigo, nausea, colic, and tenesmus. There was copious vomiting of a greenish coloured matter, with thirst, dilated pupils, stertorous respiration, convulsions, and tetanic stiffness of the limbs. One child died in the acute stage; the others died apparently from secondary consequences during treatment. (Orfila, Op. cit. ii. 273.) From three to four berries of this plant have been found to produce sleep.

ANALYSIS.—The plants can only be identified botanically by an examination of the leaves and berries. The active principle in both is an alkaloid, SOLANIA, which is itself a poison, although not very energetic. Two grains of the sulphate killed a rabbit in a few hours. According to Liebig, this poisonous alkaloid is formed in and around the shoot of the common potato, when it germinates in darkness: but there is no evidence that the potatoes are thereby rendered injurious. Their noxious properties are probably due to other causes, (see ante, p. 428.)

There are a few other vegetable substances which will here require notice. These exert a special action on the brain and nervous system, producing confusion of intellect, and deadening sensibility. The first of these is Camphor.

CAMPHOR.

I have not been able to meet with any case in which camphor has caused death in the human subject; but it has on several occasions produced rather alarming symptoms, and would probably have destroyed life, had it not been early removed from the stomach. In the few cases that have been observed, its effects were somewhat different, although both in man and animals they were referable to an impression on the brain and nervous system.

SYMPTOMS AND APPEARANCES.—The following case is reported by Mr. Hallett, of Axminster. A woman swallowed in the morning about a scruple of camphor dissolved in rectified spirits of wine and mixed with tincture of myrrh. In half an hour she was suddenly seized with languor, giddiness, occasional loss of sight, delirium, numbness, tingling and coldness of the extremities, so that she could hardly walk. The pulse was quick and respiration difficult, but she suffered no pain in any part. On the administration of an emetic, she vomited a yellowish liquid, smelling strongly of camphor. In the evening, the symptoms were much diminished, but she had slight convulsive fits during the night. The next day she was convalescent; the dyspnœa, however, continued more or less for several weeks. The dose did not probably exceed *twenty grains*,—this is the smallest dose of camphor which appears to have been attended with serious symptoms. A man, aged thirty-nine, swallowed about thirty-five grains of powdered camphor, prepared for lozenges. In twenty minutes, giddiness and dimness of sight came on; and he fell from a chair in a kind of epileptic fit, which lasted about ten minutes. The extremities were cold, the pulse was frequent and scarcely perceptible:—when roused he had scarcely power to articulate. A quantity of a clear liquid, smelling strongly of camphor, was drawn off by the stomach-pump. The man did not recover for a week, suffering chiefly from general exhaustion and suppression of urine: this latter symptom continued more or less for three months afterwards. There was no disorder of the stomach or bowels. Dr. Christison refers to a case where half a drachm of camphor given in an injection produced numbness of the scalp, and other nervous symptoms. In two other instances mentioned by him, in each of which forty grains had been taken, the symptoms were vertigo, chilliness, convulsive fits and delirium. In larger doses, symptoms of irritation make their appearance. Dr. Siemerling of Stralsund, relates that a man, aged sixty-nine, swallowed two drachms of camphor, in order to relieve some rheumatic symptoms under which he was labouring. When seen three hours afterwards, he resembled a drunken person. He complained of burning heat in the mouth, throat, and stomach,—throbbing in the head, pains in the course of the spine and a ringing in the ears,—and the appearance of a dazzling light before the eyes: these symptoms were followed by subsultus tendinum, and insensibility. In this state, he continued for an hour and a half, perspiring profusely. The man slowly recovered; but none of the camphor appears to have been ejected from the stomach. (Wildberg's Jahrbuch, 1837, 3 B. 4 H.) In a case reported in the Medical Gazette, (vol. xi. 772,)—two drachms were taken by a physician, and all that he experienced was, lightness in the head with great exhiliration. There was no derangement of the stomach or bowels. He slept profoundly for some hours, and awoke very weak and exhausted. He also perspired greatly during his sleep. It is difficult to draw any conclusion from this case, as the quantity taken was conjectural; and the patient was not seen by any person, while labouring under the effects of the poison. M. Raspail has lately advocated the use of camphor in very large doses as a universal remedy for diseases. This rash practice has been in some instances attended with dangerous effects. A man who had taken in divided doses about sixteen grains in twenty-four hours, complained of a sense of suffocation, dyspnœa, nausea, and great anxiety. (Jour. de Pharmacie, Fév. 1846, 121.) In the same journal three other cases are mentioned, in which alarming effects followed the injudicious use of this drug. The largest dose of camphor that has been taken, was in a case which occurred to Wendt, of Breslau. Eight scruples were swallowed by a drunkard, dissolved in spirit. The symptoms were vertigo, dimness of sight, delirium, and burning pain in the stomach. There was *no vomiting:* the man recovered! This case shows, that camphor cannot be regarded as a very active poison. (Wibmer, op. cit. iii. 212.) In Orfila's experiments on animals, the mucous membrane of the stomach was found inflamed (ii. 493.)

Treatment.—The free use of emetics.

Chemical analysis.—The camphor would probably be found in the state of lumps, or dissolved in spirit. No difficulty would occur in identifying this substance, except perhaps in a case where it had proved fatal and existed in the contents of the stomach. Its presence would, however, be immediately known by its powerful and peculiar odour, which has been perceived throughout the whole body in dogs poisoned by it. If it were diffused in the form of lumps or powder, these might be easily separated from the contents, owing to the great insolubility of this substance. In general, it might be expected that some portions would float to the surface of water, in which it is very insoluble. In a doubtful case, the solid contents of the stomach should be concentrated and treated with a large quantity of alcohol: the alcoholic liquor filtered, and the camphor separated by adding water. It is a white solid,—possessing a well known odour,—easily dissolved by alcohol, and again separated by water,—entirely volatile without residue, and burning with a rich yellow smoky flame.

ALCOHOL.

The only form of poisoning by alcohol, which a medical jurist has to encounter, is that which arises from the taking of large quantities of spirituous liquors—such as gin, whisky, rum, and brandy. The two last-mentioned compounds contain about fifty-three per cent. by measure of alcohol, while gin and whisky are rather stronger,—gin containing as much as fifty-seven per cent.

Symptoms and appearances.—A large quantity of spirit has been known to destroy life immediately, although such a case is rare. Orfila mentions an instance in which a man died immediately from the effects of a large dose of brandy. (Op. cit. ii. 528.) In general, the *symptoms* come on in the course of a few minutes. There is, confusion of thought, with inability to stand or walk, a tottering gait and vertigo, followed by coma. Should the individual recover from this state, vomiting and sickness supervene. This form of poisoning presents some singular anomalies:—thus the insensibility may come on suddenly, after a certain period. Dr. Christison met with a case, where the individual fell suddenly into a deep stupor, some time after he had swallowed sixteen ounces of whisky—there were none of the usual premonitory symptoms:—in another instance, a person will apparently recover from the first effects,—then suddenly become insensible, and die convulsed. Convulsions are, however, by no means a necessary attendant on poisoning by alcohol. Orfila makes their absence a ground of diagnosis between poisoning by alcohol and opium (Op. cit. ii. 530,)—and Dr. Ogston only observed them twice out of many cases: the subjects in these two instances were young. In poisoning by alcohol the supervention of the symptoms is not commonly so rapid as to prevent an individual from performing locomotion or certain acts of volition. The more concentrated the alcohol, the more rapidly are the symptoms induced, and they are then more severe in their character. Diluted alcohol generally produces the stage of excitement before stupor, while in the action of concentrated alcohol there may be profound coma in a few minutes. This appears to indicate an action by sympathy on the nervous system; as the diluted alcohol is in a condition most favourable to absorption. Alcohol may act as a poison by its *vapour*. If the concentrated vapour be respired, it will produce the usual effects of intoxication. It is generally known that persons who have been for the first time employed in bottling spirits, are easily intoxicated by the alcoholic vapour. There is a case on record in which a child two years of age, was thrown into an apoplectic stupor by the alcoholic vapour of Eau de Cologne. In this way a child might be destroyed, and no trace of the poison be found in the stomach.

Diagnosis.—It is necessary to make a distinction between the effects of alcohol, and the symptoms arising from concussion of the brain, or poisoning by opium. With respect to concussion it can only be confounded with the more advanced stage of poisoning by alcohol, *i. e.* where there is profound coma. Intoxication may in general be easily distinguished by the odour of the breath, for so long as the symptoms last, the alcohol passes off by the lungs. If there be no perceptible odour of any alcoholic liquid, the presumption is that the symptoms are not due to intoxication. When the alcoholic odour is perceptible, they may still be combined with the effects of concussion—

a fact which can only be cleared up by a history of the case, or a careful examination of the head for marks of violence. In poisoning by opium there will be a strong smell of this drug in the breath, the symptoms come on more gradually, and are marked by stupor, passing into complete lethargy, with perfect inability to walk. In poisoning by alcohol there is either very great excitement some time before the stupor, which comes on suddenly, or the individual is found in a state of deep coma a few minutes after having taken the poison. In poisoning by opium the face is pallid, and the pupils are contracted:—in poisoning by alcohol the face especially, if there be excitement, is more commonly flushed, and the pupils are generally dilated. Another fact to notice is, that while perfect remissions are rare in poisoning by opium, the individual, in poisoning by alcohol, frequently recovers his senses and dies subsequently. When coma has supervened, the patient may be roused by a loud noise or a violent shock in either case, and it is very difficult under these circumstances to draw a well-marked distinction. The odour of the breath, or an examination of the fluid drawn from the stomach by the pump, will at once lead to a diagnosis: but the treatment is the same in both cases.

In respect to *post-mortem appearances:* the stomach has been found inflamed,—the mucous membrane having been in one case of a bright red, and in another of a dark red-brown colour. When death has taken place rapidly, there will be a strong odour of spirits in the contents; but this may not be perceived, if many hours have elapsed before the inspection is made. The brain is found congested, and, in some instances, there is effusion of blood or serum beneath the membranes. In a case, observed by Dr. Geoghegan, in which a pint of spirits had been taken, and proved fatal in eight hours, black extravasation was found on the mucous membrane of the stomach; but no trace of alcohol could be detected in the contents. (Dub. Med. Press, i. 298.) A very good account of the post-mortem appearances has been published by Dr. Nicol, of Inverness. A man, aged 26, drank a large quantity of whisky with some friends. While returning home, he appeared much intoxicated, fell, uttered a few words, and immediately became quite insensible. His companions supposing him to be drunk, carried him home and placed him in bed, where he was found dead the following morning. The body was inspected seven hours afterwards. The skin of the back and depending parts was livid; and under the angles of the jaws and along the sides of the neck, of a deep purple colour. The thoracic viscera were healthy, the gastric veins distended, and the liver congested. On opening the stomach there was an odour of ardent spirits. The mucous membrane from about half way up the œsophagus, and for about eighteen inches along the intestines, was found highly injected. The corrugated part of the mucous membrane of the stomach was of a deep crimson colour. There was general congestion of the vessels of the brain and membranes. The individual had obviously died from apoplexy, brought on by the dose of alcohol (Lond. and Ed. Mon. Jour. June, 1844.)

The *quantity required to destroy life* cannot be very well determined, as it depends on the age and habits of the party. A boy, aged seven, was killed by taking two wine-glassfuls of brandy. Death may take place in a few minutes, or not until after the lapse of several days. The shortest fatal case which I have found reported, excepting that above quoted from Orfila, was that of a man who died in half an hour after swallowing a bottle of gin for a wager. This occurred in London in 1839: in a quarter of an hour after taking the gin he appeared intoxicated;—he soon became insensible, and died in *half an hour*, although a large quantity of the spirit had been removed by the stomach-pump. In general, if the case proves fatal, death takes place within twenty-four hours. Alcohol, it must be remembered, may destroy life indirectly. *i. e.* by exciting an attack of congestive apoplexy in those who are predisposed to this disease. An instance of this mode of operation has been related above.

Cases.—The following case occurred in 1840. A boy, aged seven, swallowed about three ounces of brandy:—shortly afterwards he was observed to stagger,—he was sent to bed and vomited violently. In about four hours he got up and sat by the fire; his head, face, and neck were very red, and he was in a profuse perspiration. Half an hour afterwards he was found perfectly insensible, strongly convulsed, and the skin cold. He died in about thirty hours.

As a remarkable contrast to this case may be mentioned that of a little girl, aged seven, admitted into the Westminster Hospital in December 1846, who recovered from a much larger dose of an alcoholic liquid. As nearly as could be ascertained, this child swallowed *eight ounces* of *undiluted rum* during the absence of her parents from the room. Five minutes afterwards they found her lying on the floor insensible. On admission, twenty minutes after taking the rum, she was perfectly comatose,—the countenance pallid and bedewed with perspiration, pupils much contracted, extremities relaxed, pulse quick and jerking, skin cool and moist; respiration not accelerated, and scarcely perceptible. The stomach-pump was used, and the tepid water employed came away with a strong spirituous odour. The child was temporarily roused from its comatose state by cold affusion applied by means of a watering-pot. She continued in a state of insensibility for six hours, and the pupils were then much dilated. The comatose condition ceased in about eight hours after she had taken the alcohol: she was then sensible, and answered questions readily. (Medical Times, January 16, 1847, 313.) The recovery here must be ascribed to the early and energetic treatment. In some respects, it will be observed, this case resembled one of poisoning by opium.

One of the remarkable features of poisoning by alcohol, is that a *remission* of the symptoms is by no means unfrequent, and that death sometimes takes place suddenly after some hours or days, when the individual appears to have recovered entirely from the effects. The case of the boy above related, furnishes an instance of this. Another occurred to Mr. Thomas. A man, aged 26, drank after dinner, in the course of a few hours, a pint of whisky, and during the evening of the same day sixteen ounces of raw rum, one-half of which he drank at once, and the other half in three minutes. Five minutes afterwards he was found fast asleep, snoring, his mouth open, and saliva flowing from it; he became quite insensible, and fell backwards in his chair; his countenance was pale and cold, the surface cold; the breathing stertorous; there was no pulse; the eyes were half-open, and the pupils unequally dilated and insensible to light. The stomach-pump was used and a quantity of spirituous liquid drawn off. The following day he was rational, but remembered nothing of what had passed; he was feverish, the pulse rapid, and breathing quick. He complained of pain at the pit of the stomach, and vomited all that he took. On the third day he was suddenly attacked with great difficulty of breathing, and died in about an hour. On inspection there was a patch of the mucous membrane of the stomach of a cherry-red colour, and the intestines were more injected than natural. The brain and its membranes, as well as the heart, were healthy; there was effusion in the chest. (Med. Times, June 21, 1845, p. 219.) These cases are of importance, because at an inquest a jury may be erroneously led to suppose that the alcohol was not the cause of death, but that the individual had died from subsequent maltreatment.

In April 1839, a case of poisoning by gin was communicated by Dr .Chowne to the Westminster Medical Society. A boy, aged eight, was found insensible about half an hour after he had swallowed the gin. The quantity taken was supposed to have been half a pint. The liquid drawn from the stomach seven hours afterwards, had no odour of gin:—nor was the odour perceptible in the breath. He was insensible and motionless, the limbs relaxed and powerless, the face pale and the surface cold. The pulse was quick, small, and feeble. He died without rallying or recovering his consciousness, sixty-seven hours after taking the poison. On inspection, there were no well-marked appearances found in the body,—the brain was healthy:—there was slight effusion of a serous liquid, and the veins of the pia mater were distended. The stomach was pale, and free from any mark of inflammation.

Chronic poisoning.—When alcohol has been taken for a long period in the shape of intoxicating drinks, the individual suffers from a series of diseases the characters of which are well marked. The usual effects are irritation of the stomach and intestines, pyrosis, vomiting, scirrhus of the stomach, diarrhœa, jaundice, cerebral congestion, dropsy, diabetes, paralysis, *delirium tremens*, and insanity. After death morbid changes are discovered in various organs; and the liver is especially affected. This organ is commonly enlarged, and of a lighter colour than natural: it is called the nutmeg, or the drunkard's liver. It is not unusual to find the kidneys in a state of granular degeneration.

Of all the common consequences of the abuse of alcoholic liquids, *delirium tremens* is by far the most frequent. Although a result of chronic poisoning, a state analogous to it has been known to supervene rapidly, as in the following case:—A boy, five years of age, swallowed a large quantity of brandy. Vomiting speedily followed, and he passed a restless night. In the morning it was observed that he had tremor of the hands, and that he could not hold a cup steadily. Convulsions with cramps ensued. The pulse was slow; the look timid, the pupil dilated, and the countenance pale. Delirium supervened, and there was dysuria, with great thirst. Under treatment the symptoms abated, but there was a return of the tremor towards evening. An opiate was given, and the symptoms disappeared. (Gaz. des Hôpitaux, and Med. Gaz. xxxviii. 554.) Delirium tremens is commonly observed when, after long abuse, alcoholic liquids are suddenly discontinued:—it is the result of the withdrawal of the stimulus, hence the symptoms are often mitigated when the use of alcohol is resumed. Something analogous to this is observed in chronic poisoning by opium.

Treatment.—The contents of the stomach should be withdrawn by the pump as speedily as possible. Cold affusion, if the surface be warm, or, as suggested by Dr. Christison, the injection of cold water into the ears, may serve to rouse the individual. Death may take place even when the stomach has been thoroughly evacuated, but this affords commonly the only chance of saving life. Ammonia may be employed as a stimulant, and bleeding may be resorted to if there should be great cerebral congestion. Bleeding should in any case be employed with great caution, as it is apt to depress the vital powers and diminish the chance of recovery. A copious supply of tea or strong coffee may be given until the stomach can be thoroughly cleared by the stomach-pump. The electro-magnetic apparatus may be used as in poisoning by opium; but it is necessary to remember that keeping a person roused, does not aid recovery so long as the poison is allowed to remain in the body.

Analysis.—The different spirituous liquids may be recognised in the contents of the stomach by their peculiar odour; but only when death has taken place within a few hours. The contents, if acid, should be neutralized by carbonate of soda and distilled, and the product treated with fused chloride of calcium, and again distilled. Alcohol will be obtained in the receiver. It is known—1, by its odour and volatility; 2, by its inflammability—the flame burning with a pale blue light, and depositing no carbon on cold white surfaces; 3, by its power of dissolving camphor or resins; 4, by its rapid evaporation, and the sensation of cold produced on the hand; 5, by its precipitating an aqueous solution of gum. Dr. Thomson has recommended the following test:—Drop a few grains of bichromate of potash into the vessel containing the solution to be examined, and add a few drops of oil of vitriol. If alcohol be present, even, he states, only in the proportion of a drop to half an ounce or an ounce of water, green oxide of chrome will be set free, and the odour of aldehyde will be perceived. (Monthly Jour. Med. Science, Dec. 1846, p. 412.) I have not found this test so satisfactory as Dr. Thomson represents. The mixture requires to be boiled,—the change of colour is only slowly brought out, and is liable to be concealed by the intense orange-red colour of the bichromate. If the alcoholic liquid be coloured the effect may not be perceived. I have found that formic acid produces precisely similar results where no alcohol is present. It is important to remember that alcohol may exist in the contents of the stomach, although the odour of it may not be perceptible—in some instances it may be concealed by other strong odours. In all cases the contents should be distilled. It would not be safe, when the evidence of the presence of alcohol in the body was material, that a medical jurist should rest satisfied with any other evidence, than its separation by distillation, and the subsequent demonstration of its chemical properties.

Absorption.—There can be no doubt that alcohol is absorbed, although absorption does not appear to be absolutely necessary to its action as a poison. According to the late researches of MM. Bouchardat and Sandras, alcohol passes undecomposed into the blood, but it is not eliminated by any secretory organ: a small portion only escapes by the lungs, and may be collected in the gases and vapours exhaled. If the quantity absorbed be large, the arterial blood retains the colour of venous, and may give rise to asphyxia. The oxygen received into the lungs during respiration, transforms a portion of the alcohol into water and carbonic acid: but acetic acid may even be a product of intermediate conversion. Alcohol and the compounds derived from it disappear very rapidly from the system. (Comptes Rendus, 1846.) These results do not exactly accord with those of Dr. Percy, who states that he has detected alcohol in the bile and urine. The tendency of alcohol appears to be rather to diminish the secretion of urine. (Med. Gaz. xxxviii. 430.) As a proof of the diffusion of the poison by absorption, it may be stated that in persons who die from the effects of alcohol, the odour of it is often perceived in the brain or in the serum effused in the ventricles. Dr. Bradley inspected, six hours after death, the body of a man who had died from the effects of a large quantity of alcohol. About eight or ten ounces of dark fluid blood escaped from the sinuses of the brain: this organ exhibited great vascular turgescence. There was slight extravasation in the corpora striata, and some serum effused in the lateral ventricles. This serum had a strong and well-marked alcoholic odour. (North. Jour. Med. June, 1845, page 64.) Dr. Christison and Dr. Percy have in three cases separated alcohol from the brain by distillation, and the latter has succeeded in detecting it in the liver and the blood. This is more satisfactory than a reliance upon odour, as an odour of a spirituous nature may be perceptible where no alcohol exists, and where there may be no reason to presume that alcohol has been taken.

POISONING OF ALCOHOLIC LIQUIDS.—Instances have occurred in this metropolis where alcoholic liquids have been made the vehicles for administering powerful poisons, such as opium, prussic acid, stramonium, tobacco, or cocculus indicus. Persons have been thus rendered insensible; and in this state have been robbed or murdered. Such cases may commonly be recognised by the fact that the symptoms, when known, are of far too severe a character to be referable to the small quantity of alcoholic liquid taken. Tincture of opium is not unfrequently administered in this way: and in such a case there may be some difficulty in deciding whether the symptoms of intoxication be due to the drug or to the spirit.

According to Dr. Ure, the best London porter always contains opium as a fraudulent adulteration. He has found that, when diluted, it gives a browish-red colour with permuriate of iron, indicative of the presence of meconic acid, while tincture of hops gives only a greenish-coloured liquid. Having precipitated porter by acetate of lead, he found, on decomposing this precipitate by sulphuretted hydrogen gas, that he obtained clear evidence of the presence of meconic acid; but he did not succeed in discovering morphia. (Med. Gaz. vi. 73.) These facts it may be proper for a medical jurist to bear in mind when called upon to investigate charges of administering opium in porter; but in repeating Dr. Ure's experiments upon various specimens of London porter, I have not obtained any results indicative of the presence of opium in this liquid. The precipitate obtained on adding a solution of acetate of lead to eight fluid ounces of porter, yielded not the slightest trace of meconic acid. It appears highly probable that porter is occasionally adulterated by the retail-dealers with an extract of cocculus indicus. A friend informed me that he traced singular effects, resembling those caused by a weak infusion of this poison, to a large number of families who were supplied with porter from one particular house! Unfortunately there are no satisfactory means of detecting by chemical processes this nefarious adulteration. The object of the fraud is to give apparent strength to a poor and diluted liquid by conferring upon it stupefying qualities. The effect produced, is, however, widely different from that caused by alcohol:—there is dizziness, with occasional confusion of thought—complete powerlessness, and a strong tendency to sleep at intervals in a half-waking state.

ETHER.

General Remarks.—The effects produced on the system by the administration of Sulphuric, or any other form of ether, are not unlike those occasioned by alcohol. Orfila found that about half an ounce of sulphuric ether, administered to a dog, caused, in a few minutes, a disposition to vomit. This was followed by vertigo, and in ten minutes by an entire loss of power in the muscles. Respiration was painful and hurried, but there were no convulsions. After a slight abatement in the symptoms, the dog fell into a state of insensibility and died in three hours. The whole of the mucous membrane of the stomach was of a blackish-red colour, and with the other coats intensely inflamed. There was slight inflammation in the duodenum; but the rest of the alimentary canal was in a healthy condition. The heart contained black blood partly coagulated: the lungs were gorged with fluid blood. (Op. cit. ii. 531.)

SYMPTOMS AND APPEARANCES.—Very little is known concerning the action of large doses of *liquid* ether taken into the stomach. It has, in moderate doses, a hot burning taste, and produces during swallowing a sense of constriction in the throat. It causes like alcohol great excitement and exhilaration, with, subsequently, intoxication, but persons may become habituated to it;

and thus after a time it may be taken in very large quantities with comparative impunity. The medicinal dose is from half a drachm to two drachms. Dr. Buchanan has known seven drachms of it taken at once: it produced at the pit of the stomach, a most uneasy sensation of heat and pain, which only the callous stomach of a dram-drinker could stand. (Med. Gaz. xxxix. 715.) In 1845, a young man was brought before one of the London Police-magistrates, in a stupefied state: to those present he appeared to be intoxicated. It was proved in evidence that he was in the habit of taking ether, and that he was then labouring under its effects. It appears that he frequented the shops of druggists, and swallowed this liquid in large doses. There is no instance reported of ether having caused death when taken in the liquid form: but it has never been swallowed at once in the same excessive doses as alcohol. It does not admit of dilution with water to the same degree as alcohol, and therefore it acts, cæteris paribus, as a more violent local irritant. It requires ten parts of water to dissolve one of ether: hence, unless, as Dr. Buchanan has remarked, the water be in very large proportion, it does not render the ether palatable to most persons. It is at present impossible to give any precise opinion respecting the smallest quantity of this liquid which would destroy the life of an adult.

Effects of ether-vapour.—The recent introduction of the vapour of sulphuric ether for the purpose of producing insensibility during the performance of surgical operations, renders it necessary to make a few remarks upon this form of poisoning. It has been long known that the vapour of this liquid acted on the system as a powerful narcotic. Orfila mentions the case of a young man who was thrown into a state of insensibility by reason of his having respired ether-vapour. He remained for several hours in an apoplectic condition, and would have died but for his removal to fresh air and the application of proper means for his recovery. (Toxicologie, éd. 4ème. 1843, ii. 532.) Dr. Christison quotes a similar case where a female was found lying in her bed quite dead, in consequence of her having respired, during the night, an atmosphere strongly charged with ether-vapour. On inspection the stomach was found reddened internally, and the lungs were gorged. (Op. cit. 965; also, Ed. Med. and Surg. Journal, xxxv. 452.) The poisonous effects of the vapour have been therefore known for a long time, although the attention of the profession has been only of late particularly drawn to the subject.

Ether it is well known gives off a heavy vapour, (sp. gr. 2·58,) which possesses a strong odour at all temperatures. It is exceedingly diffusible and volatile, properties which are more favourable for the operation of this liquid in the state of vapour, than for the action of alcohol. When the vapour is respired, it enters the blood in the pulmonary vessels, and the effects are almost immediate. The individual falls into a lethargic condition, the respiration becomes slow, deep and loud, the skin pale and cold, the lips assume a darker hue, the pulse is quickened, the eye is glassy and the pupil dilated: the whole body is flabby and relaxed. A small quantity of ether introduced into the blood through the lungs, produces these striking symptoms in from two to four minutes: and if fresh air be substituted as soon as unconsciousness begins, they disappear just as rapidly. In a more advanced stage the pulse slackens, and the temperature of the body rapidly falls. Half an ounce of ether, or even less, inhaled in the form of vapour, would produce a much more powerful effect on the system, than one or two ounces taken into the stomach as a liquid. The sudden cessation of the symptoms, and the restoration of sensibility, are owing to the rapid elimination of the vapour through the lungs. If the respiration of the vapour be prolonged for from ten minutes to half an hour, there is coma, the pulse sinks, and there is some difficulty in rousing the individual. The after-effects are also more serious,—there is exhaustion, a feeling of stupefaction, with other unpleasant narcotic symptoms: but occasionally the patient has fallen into a quiet sleep. The most remarkable effect in those who suffer under this form of poisoning, is the apparently complete paralysis of the nerves of sensation; for the most painful operations have been often borne by persons in this state without any consciousness of pain. In some instances, unpleasant sensations are stated to have been experienced, probably the result of association, for no consciousness whatever has existed that an operation had been performed. Another singular fact is, that although there is a general relaxation of the limbs, there is still a power of moving them, and the senses have been preserved while general sensation has been lost. It is also remarkable that the involuntary muscles do not partake of this relaxation. The vapour has varied in its effects according to idiosyncrasy and other conditions. Some persons appear to have suffered no particular symptoms: it has failed to throw them into a lethargy. This has probably arisen from its having been imperfectly respired: others have suffered from irritation of the lungs, some have become intoxicated, and others, again, have become so excited by it, as to require forcible confinement. In young subjects, nausea and vomiting have been noticed among the symptoms. As a general rule no dangerous effects appear to have followed the respiration of this vapour for surgical purposes; but this inference has been chiefly drawn from those cases in which it had been administered for a very short period; and probably there was no tendency to congestion of the brain or lungs. In cases of prolonged respiration of the vapour, serious symptoms, and even death, have resulted. Dr. Bigelow found that a young man who respired ether-vapour for thirty-five minutes, nearly sank under the effects. The pulse fell, the respiration became slow, the skin cold, and there was the most perfect insensibility. Cold affusion was employed for ten minutes without effect, and the ambulatory treatment, adopted in poisoning by opium, was then resorted to. In about half an hour he was able to lift up his head; but he did not recover his consciousness for an hour. (Lancet, Jan. 2, 1847, p. 7.) As in the case of all aerial poisons, the protracted respiration of ether-vapour must tend to render recovery difficult, by thoroughly impregnating the blood with the poison. Large dogs have been observed to lose the power of sensation in eight minutes: and they died if the action of the ether was continued for forty-five minutes. On examining the bodies of animals thus poisoned, the principal appearances have been great congestion of the vessels of the pia mater and of the sinuses of the brain, the substance being but little altered. The vessels of the medulla oblongata have been observed to be especially distended with dark-coloured blood. Both sides of the heart have been found similarly distended with dark blood; the liver and kidneys gorged; the spleen not congested: the blood black and liquid throughout the body. The cause of death therefore, may be assigned partly to the want of aeration of the blood by oxygen, and its accumulation in this state in the brain; and partly to a directly poisonous action of the absorbed vapour only manifested by its employment for a long period. In order to prevent this, it has been advised to allow the patient to breathe air occasionally, and to alternate the respiration of pure air with that of the vapour; but, unless there is a complete restoration of sensibility and consciousness, the poison must go on accumulating in the system, and if the individual be allowed to recover thus completely, it may be regarded as a commencement of its poisonous action de novo:—if not thus allowed to recover, he is in danger of sinking under its effects. The continual exhibition of morphia or strychnia, at intervals so short as not to allow of a recovery from each successive dose, must cause an accumulation in the system, and lead to fatal results. It is so with ether-vapour, and experience now points to the propriety of withdrawing its use altogether, in those cases in which the administration of it would require to be protracted for a long period. The fact that hundreds have recovered without ill effects during its temporary employment for the extraction of teeth, or similar operations, has of course no bearing on this question. A man may breathe a mixture of carbonic acid or sulphuretted hydrogen with air for a few minutes: but he would die if he was compelled to respire it for half an hour or longer.

The vapour is so insidious in its effects that it may be respired during natural sleep, without rousing the individual, and there is no doubt that it might thus be used as a ready means of destruction for the young and the aged. (Gaz. Méd. Sept. 11, 1847, p. 725.)

An interesting case has been communicated to the Medical Gazette by Mr. Nunn, by which it is rendered probable that the death of a man was occasioned by the respiration of the vapour at intervals, for a period of only *ten minutes* during an operation. He recovered from the comatose effects; but there was no tendency to reaction, and he gradually sank, and died on the second day. It was remarked in this case that there was great flaccidity and general relaxation of the muscular system, and the arteries which were divided during the operation (lithotomy) appeared to have lost all their contractile power. On inspection, there was membranous congestion of the brain; the lungs were posteriorly engorged; the heart was flaccid of a natural size, and nearly empty; the left kidney pale, the right congested. The blood was perfectly fluid. Mr. Nunn attributed death to the effect of the vapour, and this appears to have been the most probable cause. (Vol. xxxix. 414.) A similar case has been privately communicated to me by an eminent London surgeon, in which he stated that there could be no doubt of the vapour having been the direct cause of death. The patient sank after the operation, under symptoms which in similar circumstances he had never before witnessed. In the same journal there is reported another case which became the subject of a coroner's inquest. The vapour was in this instance administered to a female, for a period of thirty-five minutes. She recovered her senses, but did not rally from the operation. She complained of numbness in the feet and legs, and the secretions were suspended. She died the following day. On this occasion the vapour appeared to induce a perfect state of paralysis of the brain and nervous system. On inspection the thoracic viscera were healthy; the lungs were slightly congested posteriorly; the heart flabby, and containing less blood than usual; the brain healthy, its membranes rather congested; the blood generally in a liquid state. All the medical witnesses agreed, that in their judgment, there was nothing about the wound, or in the performance of the operation, to account for death; and that this was entirely due to the effects of ether-vapour (p. 585.) In another fatal case reported by Mr. Eastment, there was no disease, nor any particular state of the body to account for death. Amputation was performed for compound fracture of the thigh; the ether was inhaled for about ten minutes. After the operation the patient was not only greatly exhausted, but in a state of apparent intoxication. There were alternate manifestations of excitement and depression of the sensorial powers, at one time resembling delirium, at another syncope, and again passing into violent intoxication, until the patient, a boy of eleven years, died three hours after the operation. (Med. Gaz. xxxix. 682.) The symptoms were here such as might be expected from the poisonous effects of ether, and unlike those which usually attend collapse from an operation.

These facts, then, show that the respiration of the vapour, even for so short a period as ten minutes, may be in some instances attended with fatal consequences. Whether the vapour was properly administered or not, is, in relation to legal medicine, not so much the question as whether it caused death! In any case the inhalation of this vapour must be looked upon as temporary poisoning, with, cæteris paribus, a better chance of recovery than exists in most other instances of aerial poisoning.

CHEMICAL ANALYSIS.—Ether is at once identified by its powerful odour, even in the smallest proportion. 1. It is highly inflammable, and burns with a yellow smoky flame. 2. When shaken with its bulk of water, only a small portion is dissolved, the rest floats on the surface. If taken in the liquid form, it may be separated from the contents of the stomach by distillation, and the product rectified by redistillation with carbonate of potash. *Hoffman's Liquor* is a mixture of alcohol and ether. This may be easily examined by agitating it with half its bulk of water; the ether (beyond about one-tenth of the quantity of water used) rises to the surface and may be drawn off by a pipette. The alcohol is dissolved and retained by the water; this liquid may be procured by distillation with carbonate of potash or fused chloride of calcium, and its properties then tested.

Absorption.—Whether ether be taken in the form of liquid or administered as vapour, it is equally absorbed and circulated with the blood; in the latter state, with very great rapidity. M. Amussat noticed in his experiments on animals, that after prolonged inhalation the arterial blood had lost its red colour, and become black. The bright arterial tint was, however, soon resumed on suspending the process. Ether, besides rendering the blood black, causes it to become more liquid. The change in this fluid is very much like that which is observed in fatal cases of asphyxia. (Gaz. Méd. Sept. 11, 1847, p. 725.) M. Lassaigne ascertained by experiment, that after inhalation of the vapour there was a very great increase in the proportion of serum. Ether has been separated from the blood by M. Flandin, and it is easily perceptible in the breath, when the individual survives, even for so long a period as three or four days after it has been inhaled. (See ante, p. 29.) There is no doubt that it penetrates into the softest organs and all the secretions, and when used as a poison it would probably be detected by its odour in the body for some days after death. It is a remarkable fact, as indicative of its absorption and diffusion, that it produces its narcotic effects, when administered as a vapour by the rectum, without the production of those distressing symptoms which often accompany the first attempts at respiring it. (See Med. Gaz. xxxix. 950.)

The ready absorption and diffusion of the vapour requires notice in reference to its employment in the practice of midwifery, *i. e.*, where it is used to produce unconsciousness to the pains of delivery. The vitiated blood must of course reach the fœtus, and it is a question whether it might not act injuriously upon it, like ergot, (see ante, p. 484,) or any other absorbable poison. Accoucheurs have hitherto confined their observations to its beneficial effects upon the mother: it remains yet to be determined whether it may not produce injury to the child, and thus increase the number of still-births. Dr. Roux states, as the result of his experience, that in general the child does not suffer from any stupefaction as the result of etherizing the mother. (Gaz. Med. Oct. 9, 1847, 806.) The statistics of still-births in etherized and unetherized deliveries, might throw an important light upon this question.

[Some judicious remarks on the danger resulting from this indiscriminate use of ether-vapour in surgery, and on the serious effects which it has produced in several well-marked instances, will be found in Chelius's Surgery, Am. ed., Vol. III. p. 767.]

NARCOTICO-IRRITANT POISONS.

CHAPTER XLI.

GENERAL REMARKS ON THE NARCOTICO-IRRITANT POISONS—ANALYSIS—TREATMENT—COCCULUS INDICUS—PICROTOXIA—CORIARIA MYRTIFOLIA—DARNEL-GRASS—FOXGLOVE—DIGITALIA—BLACK AND WHITE HELLEBORE—VERATRIA—COMMON HEMLOCK—CONIA—WATER-HEMLOCK—HEMLOCK WATER-DROPWORT (ŒNANTHE CROCATA)—PHELLANDRIUM AQUATICUM—ÆTHUSA CYNAPIUM—IPECACUANHA—EMETINA—LABURNUM—LOBELIA—MEADOW-SAFFRON—COLCHICINA—MONKSHOOD—ACONITA.

General remarks.—Some remarks have been already made on the Narcotico-irritant class of poisons (ante, p. 39.) Toxicologists are not agreed respecting the distinction between these and narcotic poisons: the only difference commonly admitted is that the narcotico-irritants have a direct action on the spinal marrow and nerves, indicated by paralysis and convulsions, while the narcotics specially affect the brain: but this can scarcely be regarded as a sufficient ground of distinction, since there is a greater difference between the physiological action of Nux vomica and Belladonna, than between Belladomma and Hyoscyamus. So again Opium and Prussic acid affect the spinal marrow and produce convulsions. The whole of the narcotic and narcotico-irritant poisons might be arranged under one class, as NEUROTIC poisons, from their chief action being on the nervous system; but I have thought it advisable to retain the division which has been of late years uniformly adopted in this country.

The Narcotico-irritant poisons are derived from the vegetable kingdom. Their *effects* on the body are of a mixed character, since both the brain and alimentary canal are liable to be affected by them.

In order to prove fatal, they require to be exhibited commonly in large doses. The symptoms in most cases appear in about an hour; but sometimes they may be delayed for many hours. This has been especially noticed with regard to poisonous mushrooms. The symptoms commonly observed are vertigo, coma, delirium, paralysis or convulsions. Such at least are the effects resulting from Monkshood (Aconite) and Deadly Nightshade (Belladonna.) These poisons have in general a strong and well-marked taste, so that they cannot be criminally administered without suspicion being excited, or without detection. Murder by Monkshood has been accomplished by the criminal substitution of the leaves of this plant for other vegetables at a meal.

The Strychnos tribe including Nux Vomica, has a specific action on the spinal marrow, producing tetanus and convulsions, but rarely coma or delirium. Squills and Foxglove (Digitalis) produce symptoms of narcotism, *i. e.* they affect the brain; but these symptoms are commonly preceded by vomiting, with violent pain in the stomach and bowels, indicative of an irritant action.

Thus, then, there is great variety in the effects produced by this class of poisons, and the same may be said of the post-mortem appearances in the bodies of those who have been killed by them. In some instances the stomach and intestines are inflamed: in others not. Where the person has died under symptoms of narcotism, traces of cerebral congestion are occasionally found; but cases of fatal poisoning by these vegetable substances are so rare, that we have yet much to learn respecting the morbid changes which they produce.

Orfila and other toxicologists have remarked that the narcotic and irritant effects of these vegetable poisons seldom appear in the same case. The symptoms are those either of narcotism or irritation, and they sometimes alternate: when taken in *large* doses, they seem to act principally as *Narcotics;* in *small* doses, as *Irritants.*

Analysis.—Most of the narcotico-irritant poisons owe their deleterious effects to the presence of an alkaloidal principle similar to morphia, and susceptible of insulation by complex chemical processes. There is, however, considerable difficulty in extracting these alkaloids from the respective vegetables; and when extracted, the chemical differences among them, in respect to the action of tests, are very slight. Indeed, better evidence of the poisonous nature of a suspected liquid would commonly be derived from the exhibition of a portion of it to animals, than from the application of chemical tests. In a medico-legal point of view, there are, with few exceptions, no chemical tests for these poisons, when they are mixed up with organic liquids, upon which reliance can be placed. When the vegetable has been used, either in the shape of seeds, leaves, berries, or root, then valuable evidence may be sometimes procured by searching with or without the aid of a good microscope for the botanical characters of the plant; these parts of the plant, from their indigestible nature, may be found in the vomited matters or evacuations during life, or in the alimentary canal after death. The broken leaves may be separated by washing as they are quite insoluble in water: they may be therefore easily collected, dried on mica and examined by the microscope, which, under the hands of a skilful botanist, may thus reveal the nature of the poison. This source of evidence will, however, often fail, owing to the poison having been taken, in the form of extract, infusion or decoction, or even, in some instances, owing to the digestive action of the stomach itself on the vegetable matter. The active alkaloidal principle is no doubt absorbed in all cases of poisoning; but it has not yet been satisfactorily detected by chemical processes in the blood or secretions.

Some years since, I was consulted in a case in which there was hardly a medical doubt that the life of a person had been destroyed by the decoction of a narcotico-irritant vegetable. The fact, however, could not be clearly established. It is much to be regretted, that post-mortem examinations are not enforced as an indispensable part of a coroner's inquest, in all instances of narcotico-irritant poisoning. There is no department of toxicology so defective as this; only a few pathological characters have been observed in cases derived almost exclusively from foreign authorities; and in regard to the effects of some of these poisons on the human body, nothing whatever is known except that they destroy life. The acquisition of any sort of medical experience on these points, in England, is unfortunately left to be a matter of the purest accident; and yet on a trial for murder by any of these poisons, our law-authorities would expect that a witness should be perfectly conversant with their effects on the body, while the only possible source of acquiring such knowledge in a satisfactory manner, is entirely cut off from the medical profession! Some well-informed coroners have endeavoured, in performing their duties, thus to benefit the public; but the generality of them act on the principle that the inquest in such cases is merely to record the fact of death from an *external* view of the body.

Treatment.—The treatment of a case of narcotico-irritant poisoning consists in promoting early vomiting by emetics, or in drawing off the contents of the stomach by the stomach-pump. If there should be reason to suppose, from the seat of pain, that the poison has descended into the bowels, then laxative enemata may be used. Recoveries have taken place when the poison has been thus removed, even although formidable symptoms had set in. Cold affusion, or stimulants, may occasionally be required: the patient, if inclined to sleep, should always be kept roused. There is no certain chemical antidote to any of these poisons. Tannin precipitates all the alkaloids: hence it has been strongly recommended as an antidote. No injury can follow its exhibition: and a decoction of black tea will be a good substitute for oak-bark or galls. Coffee may be used as a stimulant. With respect to electricity, Ducros found that the negative current was beneficial to animals poisoned by strychnia or brucia; while the positive current produced convulsions, and accelerated death. (Canstatt, Jahresbericht, 1844, v. 297.) The narcotico-irritants appear to have no corrosive properties:—some of them give rise to a sense of burning heat in the throat and stomach,—this is a local action entirely independent of chemical change: it is especially witnessed in the case of monkshood. For the convenience of reference, the most important of these substances will be considered in an alphabetical order.

COCCULUS INDICUS.

This is the fruit or berry of the **Menispernum Cocculus**, *Linn.* (*Levant Nut,*) imported from the East Indies. It contains from one to two per cent. of a poisonous alkaloid (*Picrotoxia*). The seeds, in powder or decoction, give rise to nausea, vomiting, and griping pains, followed by stupor and intoxication. There are, so far as I am aware, only two well-authenticated instances of this substance having proved fatal to man. Several men suffered from this poison in 1829, near Liverpool: each had a glass of rum strongly impregnated with Cocculus Indicus. One died that evening; the rest recovered. (Traill's Outlines, 146.) Of the second case, the following details have been published. A boy, æt. 12, was persuaded by his companions to swallow two scruples of the composition used for poisoning fish. It contained Cocculus Indicus. In a few minutes he perceived an unpleasant taste, with burning pain in the œsophagus and stomach, not relieved by frequent vomiting,—as well as pain extending over the whole of the abdomen. In spite of treatment, a violent attack of gastro-enteritis supervened, and there was much febrile excitement, followed by delirium and diarrhœa, under which the patient sank on the nineteenth day after taking the poison. On inspection, the vessels of the pia mater were found filled with dark-coloured liquid blood. There was serous effusion in the ventricles of the brain, and the right lung was congested. In the abdomen there were all the marks of peritonitis in an advanced stage. The stomach was discoloured, and its parietes thinner and softer than natural. (Canstatt, Jahresbericht, 1844, v. 298.)

London porter and ale are considered, and in some instances with propriety, to owe their intoxicating properties to a decoction, or extract of these berries, a fraud not readily susceptible of detection. Cocculus indicus is sometimes used by robbers to intoxicate their victims, and to this form of intoxication, the term hocussing is applied. This substance is applied to no useful purpose whatever, either in medicine or the arts; and, under a proper system of medical police, its importation would be strictly prohibited.

Cocculus indicus is sometimes used for the purpose of taking fish. As it destroys them by poisoning them, it becomes a question whether the fish thus killed, can be safely eaten as food (see p. 140, ante.) M. Chevalier has examined this subject; and the conclusion to which he has come is, that, although instances may be cited of the fish being used with impunity as food, yet it must be regarded as a dangerous practice. The result depends on the quantity of poison used; thus, when from ten to fifteen grains of the berries were employed, and the fish was afterwards given to animals, the noxious effects were as strongly marked as if they had swallowed the poison. (Ann. d'Hyg. 1843, i. 343.)

The shell appears to act like an emetic, while the kernel, which alone contains picrotoxia, is the seat of the poison.

From the facts collected by Wibmer (Arzneimittel. *Menispermum*,) neither cocculus indicus nor picrotoxia, which forms 1-100th part of the kernel, appears to be possessed of very active properties upon large animals. Orfila gave 3·38 grains of picrotoxia to a good-sized dog: it produced frequent vomiting, but the animal soon recovered. (Toxicologie, ii. 501.)

PICROTOXIA is seen in fine white crystals, which have an intensely bitter taste. It is not very soluble in water, even on boiling it; but it is dissolved by alcohol and ether. Caustic potash dissolves it, forming a yellow solution, but it is not very soluble in acetic acid. Sulphuric acid turns the crystals of a deep brown colour, slowly changed to a deep green on adding a few drops of a solution of chromate of potash. Strong nitric acid dissolves picrotoxia, but produces in it no change of colour. There are no chemical processes by which the presence of this poison in an organic liquid can be demonstrated.

CORIARIA MYRTIFOLIA.

The leaves and berries of this plant (*Myrtle-leaved Sumach*) have a poisonous action, not unlike that of Cocculus Indicus. The leaves so closely resemble those of senna, that they are often employed as a fraudulent adulteration,—a practice likely to lead to dangerous consequences. Wibmer quotes the case of a man and his wife, who drank a decoction of senna-leaves mixed with those of the Coriaria. In a few minutes the man suffered from violent colicy pains in the abdomen, convulsions, and trismus. He died in four hours. The woman suffered from similar symptoms, but recovered. On inspecting the body of the man, the stomach was found inflamed, and of a reddish-brown colour. A portion of the decoction given to dogs killed them with similar symptoms, and on dissection the alimentary canal was observed to be inflamed. (Op. cit. *Coriaria.*) From these facts it would appear that this vegetable poison has a local irritant action.

The same question has been raised in reference to the Coriaria, as with respect to Cocculus Indicus, *i. e.* its power of giving poisonous properties to the flesh of animals feeding on it. A case in which the whole of the members of a family were thus indirectly poisoned, is elsewhere related (ante, p. 140.)

Four cases of poisoning by the berries of RHUS CORIARIA are reported in the Journal de Chimie Méd. (Avril, 1847, 197.) Two of these proved fatal. The subject of one, a child aged seven years, was found in about seven hours in a state of complete depression, with subsultus tendinum, loss of vision, and the jaws spasmodically closed: death took place the following day. In the other fatal case, the principal symptoms were very similar, with the addition of great dilatation of the pupils, and suppression of urine. The recovery of two of the children was to be ascribed to early and copious vomiting.

DARNEL GRASS.

The effects produced by the seeds of the bearded darnel, LOLIUM TEMULENTUM, when mixed with grain, have been already described. (See ante, p.

430.) There is no doubt that the noxious effects of fresh bread are frequently due to the accidental presence of this vegetable poison. It is not likely, however, to occasion injurious symptoms, unless the bread be habitually used for a long period. From experiments on animals, and from a few observations on man, it appears that the seeds of darnel, whether taken in powder or in decoction, have a local action on the alimentary canal, and a remote action on the brain and nervous system. There is heat, with pain in the stomach, accompanied by nausea, vomiting, and diarrhœa. These symptoms are followed by languor, loss of vision, ringing in the ears, and vertigo. In order to produce such serious effects, the poisonous grain must be taken in a somewhat large dose. So far as I can ascertain, there is no instance recorded of its having proved fatal to man; and as much as three ounces of a paste of the seeds has been given to a dog, without causing death. (Wibmer, op. cit. *Lolium.*)

Analysis.—The botanical characters of the plant. Pfaff has lately examined darnel, in order to discover a poisonous alkaloid; but there was no trace of such a substance. By distillation with water he obtained two kinds of ethereal oil, one lighter and the other heavier than water; they were colourless, but had the odour of fused oil.

FOXGLOVE.

Purple Foxglove (Digitalis purpurea) is a well known hedge-plant, growing abundantly in the southern parts of England. All parts of this plant, whether in the form of powder, extract, tincture, or fusion, are poisonous, and exert an action both on the brain and alimentary canal. The leaves appear to produce the most powerful effect, and they retain their noxious properties when dried. One of the best-marked cases of poisoning by foxglove, became the subject of a criminal trial at the Old Bailey in Oct. 1826. A quack was indicted for the manslaughter of a boy under the following circumstances. He prescribed for a trivial complaint, six ounces of a strong decoction of digitalis. The boy was soon attacked with vomiting, purging, and severe pain in the abdomen. After some time he became lethargic, and slept for several hours; in the night he was seized with convulsions: the pupils were dilated and insensible; the pulse slow, small, and irregular; coma followed, and the boy died twenty-two hours after taking the poison. On inspection, the membranes of the brain were found much injected, and the mucous lining of the stomach was partially inflamed. The prisoner was acquitted of the charge, because he had only given his advice on the application of the friends of the deceased! (Ed. Med. and Surg. Journal, xxvii. 223.)

The following case of poisoning by an infusion of foxglove is reported by Mr. Wilson of Leeds. (Med. Gaz. xxxiv. 659.) A healthy robust young man, affected with sore-throat, was advised to take "*throatwort tea.*" Having filled a quart pitcher with the fresh leaves of the digitalis purpurea, he poured upon them as much boiling water as the pitcher would hold. Of this strong infusion he took a teacupful on going to bed, which caused him to sleep soundly. In the morning he took a second cupful (the infusion being much stronger,) and he then went to his employment. He soon felt dizzy and heavy, began to stagger, lost his consciousness, and at length fell down in a state of syncope. On being conveyed home he vomited severely, and suffered extreme pain in the abdomen. When visited, he was conscious, complained of great pain in his head; the pupils were dilated, and the surface was cold, pallid, and covered with a copious perspiration. The pulse was low, about 40 in the minute,—three or four feeble pulsations being succeeded by a complete intermission of several seconds; and each stroke, though weak, was given with a peculiar "explosive shock." There was still great pain in the abdomen, with incessant and violent vomiting, no diarrhœa,—suppression of urine, and an abundant flow of saliva. Brandy and ammonia with warmth were employed, and after reaction had commenced, purgatives were administered. The man slowly recovered, but the pulse presented its peculiar beat and weakness for several days; and during this time the man could not bear the upright position. This case proves that salivation may be produced by the plant.

In another instance, a young man swallowed a strong decoction of foxglove by mistake for purgative medicine. He was soon seized with vomiting, pain in the abdomen, and diarrhœa. In the afternoon he fell asleep. At midnight he awoke, was attacked with violent vomiting, colic, convulsions, dilated and insensible pupils; and his pulse was slow and irregular. He died twenty-two hours after taking the poison. (Wibmer, op. cit. *Digitalis.*)

A few grains of the powdered leaves have been known to produce giddiness, languor, dimness of sight, and other nervous symptoms. A drachm has, however, been taken without causing death; but in this instance it produced the most violent vomiting. As indicative of the singular effect of this poison on the nerves of sensation, it may be stated that a coal-fire appeared to the patient to have a blue colour. A common effect of this poison is great depression of the heart's action.

The post-mortem appearances produced by foxglove are an inflammatory condition of the stomach and intestines, fluidity of the blood, and loss of irritability of the heart.

In the case of a man, æt. 50, the tincture, taken in medicinal doses for about twenty days, produced the following train of symptoms. (Med. Gaz. xxxi. 270.) The pulse, which, during a former use of the medicine, had lessened by ten or fifteen beats in a minute, sank almost to half its usual number. The patient was tormented by the most painful disquietude, so that, even in the night, he left the bed every moment, could not sleep, and with his eyes open conversed with persons who were not present. At the same time the pupils were dilated, the conjuctiva both of the eye and the lids was red; he had but little appetite, with great nausea, violent thirst, and dryness of the mouth; the alvine evacuations were scanty; the secretion of urine was increased. These phenomena, which obviously were merely the effects of the digitalis, had lasted six days, when the restlessness diminished, sleep returned, and the dilatation of the pupils disappeared. This case shows that digitalis possesses accumulative properties; and that it cannot be given for a long period medicinally without producing dangerous symptoms. Dr. Elliotson states that he has known persons who had been in the habit of taking this medicine for a long period die very suddenly, as if from accumulation of the poison in the system, and its fatal action on the heart.

In a case of poisoning by foxglove, in addition to the free use of emetics, vegetable infusions containing tannin should be given. According to the researches of M. Homolle, this renders the active principle—*digitalia*—insoluble.

The medicinal dose of the infusion is from half an ounce to one ounce;—of the tincture, from ten to forty minims;—and of the powder, from half a grain to one grain and a-half. The medicinal preparations vary considerably in strength, a fact which will explain why they have been administered in much larger doses than those here assigned, without producing dangerous effects. Dr. Pereira states that twenty drops of the tincture were given to an infant, labouring under hydrocephalus, three times daily for a fortnight, without causing any untoward symptom; and he has frequently prescribed, for an adult, one drachm of the tincture three times daily for a fortnight, without producing any marked effect. The tincture has been sometimes prescribed medicinally in doses of half an ounce to an ounce; and on one occasion two ounces were taken in two doses, without giving rise to the slightest inconvenience. (Mat. Med. ii. 1213.) These facts show either that foxglove is not so powerful a poison as it is commonly supposed to be, or that the proportion of digitalia is liable to great variation in the alcoholic solution.

Analysis.—When foxglove has been taken in substance, it can be identified only by its botanical characters. In the form of infusion, decoction, or tincture, and when mixed with organic liquids, there are no chemical processes by which the nature of the poison can be determined.

Digitalia.—The active principle of foxglove is called **Digitalia.** Its properties have been recently investigated by M. Homolle. (Journ. de Pharmacie, Janvier 1845, 57.) The process for obtaining it is exceedingly complex. It is a white, inodorous, imperfectly crystalline substance. It is so intensely bitter, that it gives a sensible bitterness to 200,000 parts of water; but the taste of digitalia itself is only slowly manifested, in consequence of its great insolubility. Cold water dissolves only 1-2000th, and hot water only 1-1000th part: it is much more soluble in alcohol and ether. When dissolved in either menstruum, it has neither an acid nor alkaline reaction. It does not form salts with acids. It immediately decomposes nitric acid, evolving nitrous acid vapours, and producing a rich orange-yellow coloured solution, which acquires in a few days a golden-yellow tint. Sulphuric acid at first blackens it, but subsequently forms a brownish-black liquid, which passes in a few days to a red-brown, amethyst, and finally a rich crimson colour. If a portion of the reddish-brown liquid be dropped into a small quantity of water, it gives to it a rich green colour. Muriatic acid produces with it a yellow, speedily passing to a bright-green colour. A dose of more than 1-16th of a grain could not be taken by an adult without causing symptoms of poisoning. This quantity was found to be equal in strength to about eight grains of the well-prepared powder of the leaves.

HELLEBORE.

There are several varieties of Hellebore; but the two plants which are most commonly used as poisons under this name, are the Black and white Hellebore.

Black Hellebore.—This plant, which is the **Helleborus Niger** of the modern, and **Melampodium** of the old pharmacopœias, is known under the name of *Christmas Rose*, from its flowering in January. In Lancashire it is called *Brank-ursine.* Another variety, **Helleborus Fœtidus**, sparingly grows in shady places and on a chalky soil, flowering in March and April: it is known under the names of *Bear's foot*, *Setter-wort*, *Helleboraster*.

According to Wibmer, the roots of the Black Hellebore possess the greatest activity; but the leaves are also highly poisonous when used in the form of infusion. By long boiling the poisonous properties of the plant are diminished, probably owing to the loss of the volatile principle, which is an acrid oil. The roots and leaves have a local irritant action, producing violent vomiting and purging in small doses, with severe pain in the abdomen, followed by cold sweats, convulsions, insensibility, and death. The powdered root, in a dose of a few grains, acts like a drastic purgative. In a case reported by Morgagni, half a drachm of the aqueous extract killed a man, aged 50, in eight hours. The symptoms were severe pain in the abdomen and violent vomiting. After death, the whole of the alimentary canal was found inflamed, but especially the large intestines. (Wibmer, op. cit. **Helleborus.**) A case is quoted by the same writer, in which a table-spoonful of the finely powdered root (taken by mistake for rhubarb) caused severe symptoms of irritant poisoning, which did not disappear for four hours. The man recovered on the fourth day.

The experiments performed by Orfila on animals show that this poison acts like a *local* irritant when applied to a wound on the skin. (Op. cit. ii. 369.)

Hellebore is a favourite remedy for worms with quacks and rural doctresses. It is not, therefore, surprising that it should be occasionally administered in an overdose, and cause death.

Mr. J. H. Todd, Coroner for Southampton, has kindly forwarded to me the report of an inquiry which took place before him, in Nov. 1845, in which a child under two years of age was poisoned with an infusion of hellebore, administered to it by its grandmother, for the purpose of destroying worms. The leaves of the plant (Bear's foot) were bruised, and boiling water poured over them. Two dessert spoonfuls were given to the child, who had been suffering from ague, but from which he had recently recovered. Within ten minutes after taking the mixture he was very sick, and was violently purged. The matter vomited was of a green colour, and slimy: the sickness and purging continued until the evening, when he died, *i. e.* about thirteen hours after having taken the mixture. There were convulsions before death. On inspection, the whole body appeared blanched; the eyes were sunk, and the pupils dilated. There was diffused inflammation of the mucous membrane of the stomach, and a well-marked patch of inflammatory redness, about the size of a five-shilling piece, near its centre. The small intestines, which contained a brownish-yellow fluid, were much inflamed. The cæcum contained about thirty worms. The head and chest were not examined. Death was very properly attributed by the medical witness to the action of hellebore.

The woman who prepared the infusion stated that she had frequently given it in large quantities to young children, and there were no injurious effects. It is nevertheless to be regarded as an active poison; and if persons are not always killed by such worm-medicines, it must be regarded as a very fortunate circumstance. This acrid vegetable never can be given by an ignorant person without great risk.

ANALYSIS.—The botanical characters of the leaves and roots.—Black Hellebore has a large flower with five round spreading petals, which are at first white, and afterwards become reddish-coloured, and finally greenish. The flower of Fœtid Hellebore, or Bear's foot, has five oval concave petals, of a green colour, tinged with purple at the margin.

[Two other species H. orientalis and H. viridis are equally active with the H. niger. The former is now considered to be the Melampodium of the ancients.—G.]

WHITE HELLEBORE. VERATRUM ALBUM.—The action of this plant is analogous to that of black hellebore; it is, however, more irritant and less stupefying. The powdered root produces a strong local effect, and causes violent sneezing. When taken internally, it gives rise to severe pain in the abdomen, violent vomiting and purging, followed by giddiness, dilatation of the pupils, convulsions, insensibility, and death. It produces a sense of great heat and constriction in the throat. In three cases mentioned by Dr. Pereira, in which the infusion had been swallowed, there was no purging. (Op. cit. ii. 954.)

There can be no doubt that white hellebore has all the characters of a narcotico-irritant poison. In another part of the volume, (p. 16, ante) a case has been quoted, in which, at a recent trial for criminal abortion, a medical witness expressed the opinion that it was not a poison. The numerous observations collected by Wibmer, prove that it acts most powerfully on the system. In one instance, twenty grains of the powder caused convulsions and death in three hours, and in another, a man after eating the root, died in six hours. Death was preceded by vomiting of bloody mucus, and by cold sweats. (Op. cit. VERATRUM.) The smallest quantity required to destroy life is unknown. Dr. Christison quotes a case from Bernt, in which a man took but a small quantity of the powder, and died in the course of the day. After death the same marks of irritation were found in the alimentary canal as those which have been described in speaking of black-hellebore.

ANALYSIS.—Powdered *white hellebore* root has a reddish-brown colour, resembling jalap. Nitric acid gives to it a red, rapidly passing to a dark-brown, colour. Sulphuric acid produces with it a dark-brown tint, almost black. Iodine water, a blueish grey tint, slowly brought out. The proto and persalts of iron have no effect upon it.

VERATRIA.—White hellebore owes its noxious properties to the alkaloid *veratria*, which is itself a powerful poison. A medical friend communicated tome the following fact. A physician prescribed medicinally for a lady, one grain of veratria divided into fifty pills, and three were directed to be taken for a dose. Not long after the first dose had been swallowed, the patient was found insensible, the surface cold, the pulse failing, and there was every symptom of approaching dissolution. She remained some hours in a doubtful condition, but ultimately recovered. Supposing the medicine to have been well mixed, and the pills equally divided, not more than one-sixteenth of a grain of veratria was here taken!

This proves that pure veratria is capable of exerting a very powerful effect. The common veratria of the shops is sometimes given medicinally, in doses of one-sixth of a grain. In the pure state, it forms a brownish-white uncrystalline powder, scarcely soluble in water, even on boiling, but is more readily dissolved by alcohol and ether. It has a very faint alkaline reaction, and easily combines with the acetic and other acids, forming soluble salts. It has ahot, acrid taste, without any bitterness. Strong nitric acid gives to it a light red, turning to an ochreous, colour. Sulphuric acid gives to the powder an intense orange-brown colour, which becomes of a deep claret-red on adding chromate of potash. The same colour is produced when the acid is added to the veratria dissolved in acetic acid.

[A native species, V. viride is equally poisonous, and a decoction is often used to destroy vermin. The root is very acrid. See a memoir on this plant by Dr. Osgood (Amer. Jour. Med. Sci. xvi. 279.)—G.]

CEVADILLA.

This name is given to the follicles and loose seeds of the Veratrum Sabadilla. [*Asagræa officinalis.*—G.] It is very much used as a bug-poison, and has been employed as a vermifuge: it has a strong local irritant action. It acts like white hellebore, and its poisonous properties are due to the presence of Veratria. Cevadilla also yields a very acrid alkaloid, *Sabadilline*, the effects of which on the body are unknown.

HEMLOCK.

Under this name are popularly included several varieties of plants.

COMMON OR SPOTTED HEMLOCK.

COMMON HEMLOCK, or CONIUM MACULATUM, is a well-known hedge-plant, which grows abundantly in most parts of Great Britain. Its effects on man and animals prove that it possesses active poisonous properties: these chiefly reside in the leaves and roots, and may be extracted by water. Its energy varies, probably according to season and locality. The effects produced by hemlock, have not been uniform; in some instances there has been stupor, coma, and convulsions; while in other cases, the action of the poison has been chiefly manifested on the spinal marrow,—*i. e.*, it has produced paralysis of the muscular system.

In a series of cases, quoted by Orfila, several soldiers partook of hemlock in soup. Soon afterwards, they all appeared to be intoxicated. One, who had eaten of the soup rather freely, became, in less than two hours, senseless; he breathed with difficulty; his pulse was hard, small and slow; surface cold; his face livid, like that of a person who had undergone strangulation. Emetics were administered, with temporary relief, but he became again unconscious, lost the power of speaking, and died three hours after partaking of the soup. On inspection, the stomach was found half filled with a quantity of pulpy matter, and there were some red spots on the membrane, near the pylorus. The cerebral vessels were gorged with blood, which was quite liquid. (Op. cit. ii. 427.) Dr. J. H. Bennett met with a case which illustrates the other mode of action. A man ate a large quantity of hemlock-plant, by mistake for parsley. Soon afterwards, there was a loss of power in the lower extremities: but he apparently suffered no pain. In walking he staggered as if he was drunk; at length his limbs refused to support him, and he fell. On being raised, his legs dragged after him, and when his arms were lifted, they fell like inert masses, and remained immoveable. There was perfect paralysis of the upper and lower extremities within two hours after he had taken the poison. There was loss of power of deglutition, and a partial paralysis of sensation, but no convulsions,—only slight occasional motions of the left leg; the pupils were fixed. Three hours after eating the hemlock, the respiratory movements had ceased. Death took place in three hours and a quarter; it was evidently caused by gradual asphyxia from paralysis of the muscles of respiration; but the intellect was perfectly clear until shortly before death. On inspection, there was slight serous effusion beneath the arachnoid membrane. The substance of the brain was soft; on section there were numerous bloody points, but the organ was otherwise healthy. The lungs were gorged with dark-red fluid blood; the heart was soft and flabby. The stomach contained a green-coloured pultaceous mass resembling parsley. The mucous coat was much congested, especially at its cardiac extremity. Here there were numerous extravasations of dark blood below the epithelium, over a space of about the size of the hand. The intestines were healthy, here and there presenting patches of congestion in the mucous coat. The blood, throughout the body, was fluid and of a dark colour. A portion of the green vegetable pulp was identified by Dr. Christison, as part of the *Conium maculatum*. Some of the leaves bruised in a mortar, with a solution of potash, also gave out the peculiar odour of the volatile principle CONIA. (Ed. Med. and S. J. July 1845, 169.) These two cases give a more complete account of the effects produced by hemlock upon adults, than any others yet published. It does not appear that delirium or convulsions are by any means common.

ANALYSIS.—Hemlock is known from most other plants, which resemble it, by its large, round, smooth and spotted stem. The leaves are of a dark-green colour, and smooth and shining. Every portion of the plant has a strong and disagreeable smell when bruised;—this is especially brought out when the stem or leaves are rubbed with caustic potash.

CONIA.—The active properties of hemlock depend on the presence of a peculiar volatile alkaline liquid, of a highly pungent odour, irritating to the skin, and intensely acrid to the taste. It is transparent, oily-looking, and floats on water, in which it is not very soluble. Its vapour is inflammable: it produces white fumes with muriatic acid vapour. This alkaloid, CONIA, is very soluble in alcohol and ether, and combines with diluted acids to form salts. It exists in all parts of the plant, but an alcoholic extract of the seeds contains the largest quantity. The common *Extract* of hemlock, the medicinal dose of which is two or three grains, is liable to vary much in strength, according to the mode in which it has been prepared:—when overheated, there is a great loss of conia. The presence of this alkaloid in the extract, may be readily determined by triturating it with caustic potash; if present, it is immediately set free, and may be recognised by its odour. The proportion of conia in the plant probably varies at different seasons of the year,—a fact which will account for the root having been occasionally eaten with impunity.

Dr. Christison's experiments prove that conia, whether free or combined, is a most powerful poison (ante, page 34.) Although, as it exists in hemlock, it undoubtedly operates by absorption, yet, when insulated, it destroys life so rapidly, that it must be supposed to kill, at least occasionally, without entering the blood. It produces general palsy without insensibility, and with slight occasional twitches only of the limbs of the animal. (Op. cit. 855.) It is singular that the heart does not appear to be affected by the poison, as this organ pulsates even after other signs of life have ceased. Death appears to be due to asphyxia, from the general paralysis of the respiratory muscles. A single drop of conia, applied to the eye of a rabbit, killed it in nine minutes; and three drops killed a strong cat in a minute and a half.

WATER-HEMLOCK. HEMLOCK WATER-DROPWORT.

The WATER-HEMLOCK, or CICUTA VIROSA, has given rise to several fatal accidents—its roots having been mistaken for parsnips. The whole of the plant is poisonous; but the roots are the most active, especially when gathered early or late in the year. The *symptoms* produced by the roots are vertigo, dimness of sight, headach, and difficult respiration. There is burning pain in the stomach, with vomiting, and these symptoms are accompanied by heat and dryness of the throat. Convulsions have been observed to precede death. In the cases of three children who died in convulsions from this poison, Mertzdorff found an injected state of the mucous membrane of the stomach, with redness of the epiglottis, pharynx, cardia, and pylorus: the vessels of the brain and the sinuses were filled with dark liquid blood. (Wibmer, *Cicuta*, 119.) In a fatal case which occurred to Wepfer, the patient, a man aged 20, who had eaten a large quantity of the root, was found with his face swollen and his eyes projecting. He breathed with great difficulty, and foamed at the mouth. He was seized with a severe epileptic fit: his limbs assumed a tetanic stiffness, and there was spasmodic breathing. He was quite unconscious, and speedily died. The only marked appearances were fluidity of the blood, and patches of redness on the mucous membrane of the stomach. (Wibmer, loc. cit.)

Dr. Badgley has communicated some cases of poisoning by this plant to the Montreal Medical Gazette (June 1844:)—Four children, between five and seven years of age, ate the roots of Water-hemlock by mistake for parsnips. Within half an hour, they were all seized with extreme nausea, burning pain at the epigastrium, and colicky pains in the bowels; they all complained, on reaching their homes, of sickness, for which warm milk was administered to them. Efforts to vomit were induced: in one, there was full vomiting, but in the other three nothing was ejected from the stomach. The pains gradually increased in two of them; and, in the space of about two hours from the time of their eating the roots, they were labouring under complete coma, with tetanic convulsions, the jaws rigidly fixed, profound stertor, and the whole face puffed and bloated, having precisely the appearance of the head of a person who had been for some hours under water; pulse intermitting, sometimes imperceptible. Emetics were exhibited, but without effect; and enemata of castor-oil and oil of turpentine were employed with great relief. The child who had eaten most sparingly had taken warm milk, and had vomited freely. One died in three hours; the others recovered.

Dr. Schlesier met with the following case:—A girl, æt. eight, who had eaten this plant, was found lying quite insensible. Her respiration was feeble, and rattling; the pulse soft, small, and scarcely perceptible; the pupils dilated and fixed; the face pallid; limbs flaccid: abdomen distended; and there was general coldness of the surface, with an entire loss of the power of swallowing. Stimulating embrocations and cataplasms were applied, and after some hours the pupils contracted; the body became warm; the breathing easier; but there were involuntary motions of the limbs. There was a slight return of consciousness and the power of speaking, but the difficulty of swallowing continued; and the patient died in about sixteen hours. (Canstatt's Jahresb. 1844, v. 296.)

ANALYSIS.—There are no means of identifying this plant except by the determination of its botanical characters. It grows abundantly on the borders of ditches, ponds, and streams. Its stem is thick, round, striated, smooth, sparingly branched, and often attains four feet in height. It is of a reddish colour at the branching of the umbels. The leaves are large, pinnated, and serrated: they have the taste of parsley. The root, which has a strong disagreeable smell and an acrid taste, is thick, short, hollow, and has numerous fibres at the joints. The nature of the poisonous principle is unknown.

The CICUTA MACULATA is possessed of equally virulent properties. Many fatal cases have occurred in the United States from the root having been eaten by mistake.

HEMLOCK WATER-DROPWORT. WATER-PARSNIP.—This umbelliferous plant (the ŒNANTHE CROCATA of botanists) grows on the banks of rivers, streams, and ditches. It is one of the most poisonous of the order; and it is considered to be one of the most virulent of English vegetable poisons. It is found growing abundantly in the South of Ireland. Dr. Pickells has collected thirty cases of death from the eating of the root: the quantity taken in one instance did not exceed the top of the finger in size. The symptoms were insensibility, tetanus, delirium, and insanity. Dr. Christison considers that this plant, as it grows in Scotland, is not poisonous; but there appears to be no doubt, from various recorded cases, that, as it grows in England, Wales, and Ireland, it is endowed with highly noxious properties.

SYMPTOMS AND APPEARANCES.—A very interesting set of cases of poisoning by Œnanthe has been communicated to the Medical Gazette (vol. xxxiv. p. 288,) by Mr. Bossey, of Woolwich. A number of convicts, while engaged at work, ate the leaves and roots of the ŒNANTHE. In about twenty minutes, one man, without any apparent warning, fell down in strong convulsions, which soon ceased, but left a wild expression on his countenance. Soon afterwards, as many as nine fell into a state of convulsions and insensibility. The face of the man first seized became bloated and livid; there was a sanguineous foam about the mouth and nostrils; the breathing was stertorous and convulsive; there was great prostration of strength, and insensibility: he died in five minutes. A second died, under similar symptoms, in a quarter of an hour, although the stomach-pump was used, and some leaves were extracted with the fluids. A third, who had assisted in carrying the two former, was himself seized with convulsions, and died in about an hour; and soon after him, a fourth died, in spite of the most energetic remedial treatment by cold affusion, emetics, stimulants, stimulating frictions, as well as the use of the stomach-pump. Two other cases proved fatal,—the one in nine days, and the other in eleven; and in these two cases there was irritation of the alimentary canal. On inspecting the bodies of those who died quickly, there was congestion of the cerebral vessels; and, in one instance, a layer of extravasated blood was found beneath the pia mater. In the first case, which proved most quickly fatal, the cerebral vessels were not congested. The pharynx and œsophagus had a white appearance, and contained some mucus, with portions of the root. The lining membrane of the trachea and bronchi was intensely injected with dark blood. The lungs were gorged with fluid blood. The blood in the heart was very black and fluid. The stomach and intestines were externally of a pink colour: the cavity of the stomach was lined with a thick viscid mucus, containing portions of the root. The mucous membrane was much corrugated, and the follicles were particularly enlarged. Similar appearances were met with in all. In the two protracted cases, the mucous membrane of the stomach and bowels was softened and thickened. It had a pink colour externally, but no red appearance internally. The vessels of the brain were congested. In the others, who partook of the roots, the symptoms were not so severe. Under the free use of purgatives, a considerable quantity of the root was discharged, and in a few days the men recovered. These cases show that the Œnanthe is one of the most powerful of the indigenous narcotico-irritant poisons. It destroys life with even greater rapidity than arsenic; for it here proved fatal to a strong healthy man in less than *one hour*. Chemists have not yet ascertained on what principle its active properties depend, but they appear to reside chiefly in the root.

In March 1846, Dr. Unger met with the following cases:—A woman dug up some roots which she supposed to be parsnips. They were dressed for dinner as usual in an earthen-pot in which her food was commonly prepared. The woman, as well as her husband and two children, partook of them. Dr. Unger was suddenly called to see them in the evening, and found them apparently labouring under *delirium tremens*. They were in constant motion, talking incessantly, without knowing what they said, and fancying they saw objects which had no existence. They fought with each other, and were occasionally attacked with fits of convulsive laughter. The countenance was pale; the pupil dilated; the look vague; tongue clean, moist, and tremulous; and the pulse, which, owing to the incessant motion, was felt with difficulty, appeared smaller, weaker, and slower than natural. The patients rejected every thing that was offered to them, and were obliged to be restrained by force. A neighbour who had eaten a small portion of the roots suffered from vertigo and general uneasiness: she was, however, perfectly conscious, and refused to take any remedy. Doses of sulphate of zinc were repeatedly given to the other patients, but without effect, until combined with ipecacuanha. This treatment led ultimately to the rejection from the stomach of a large quantity of the undigested root. After this, the symptoms abated; and the next morning, with the exception of a sense of weight in the head, they had all recovered. It is remarkable that there was no purging. (Gaz. des Hôpitaux, Sept. 19, 1846.) The *root* is considered to be the most active part of the plant: it is of a yellowish-white colour, and not unpleasant to the taste. A very small portion of it, unless speedily ejected from the stomach, will suffice to destroy life. The symptoms have been occasionally delayed in their appearance; but, as in Mr. Bossey's cases, when they have once commenced, they run on to a fatal termination with great rapidity.

Dr. Woodville relates, that three men ate, or rather tasted of the root. One was soon afterwards seized with convulsions, and died; two others suffered from nervous symptoms, including locked-jaw, and one of these died; a fourth had dizziness, and slowly recovered. It is remarkable that there was no vomiting, nor any tendency to vomit. The following set of cases occurred in

Ireland;—Eight boys ate the plant for water-parsnip. In four or five hours the eldest became suddenly convulsed, and died; and before the next morning, four others died. Of the remaining three, one was maniacal for several hours; the other lost his hair and nails; and the third escaped. (Medical Botany, iv. 144.) They who have vomited *early* have generally recovered.

It is not often that attempts are made to destroy others by the administration of these narcotico-irritant vegetables; but a case has recently occurred in France, in which a woman attempted to poison her husband by mixing slices of the root of this plant with his soup. His suspicions were excited by the acrid taste of the soup. The woman was tried for the crime, and M. Toulmouche deposed at the trial, that the plant from which the root had been taken, was the Œnanthe crocata,—that it was a powerful poison, and might cause death in two or three hours. The prisoner was convicted, and condemned to ten years at the galleys. (Gaz. Méd. Jan. 3, 1846, 18; also, Journ. de Chim. Méd. 1845, 533.)

ANALYSIS.—The Œnanthe crocata can be identified only by its botanical characters. It bears a greater resemblance to celery than most of the other umbilliferæ. Its stem is chanelled, round, smooth and branched, of a yellowish red colour, and growing to the height of two or three feet. The root consists of a series of oblong tubercles with long slender fibres.

FINE-LEAVED WATER HEMLOCK.

This is another umbilliferous poison, known as the PHELLANDRIUM AQUATICUM. Like the Œnanthe it is often popularly called the Water-parsnip. It grows by the banks of rivers, ditches and ponds. It is poisonous, but less virulent than the Œnanthe.

ANALYSIS.—The poisonous principle is unknown. The plant has a thick, hollow, smooth jointed stalk, usually about three feet in height; the leaves are very fine, small, and much subdivided. They are of a dark shining green colour; the root is thick, tapering, jointed, and sends off numerous long slender fibres.

FOOL'S PARSLEY.

FOOL'S PARSLEY, or LESSER HEMLOCK, is exceedingly common in gardens and hedge-rows. Its botanical name is ÆTHUSA CYNAPIUM. The leaves so closely resemble those of parsley that they have often been gathered for them by mistake. The following case of poisoning by the Æthusa cynapium is reported in a late number of the Medicinisches Jahrbuch. A woman gave two of her children some soup, in which she had boiled the root of this plant, mistaking it for parsley. They were both seized with severe pain in the abdomen, and the next morning, one of them, a boy, aged eight years, was in a state of perfect unconsciousness, and his jaws were spasmodically fixed. The abdomen was swollen; there was vomiting of bloody mucus, with obstinate diarrhœa,—the extremities were cold, and the whole body was convulsed. He died in twenty-four hours. The only appearances met with, were redness of the lining membrane of the œsophagus and trachea, with slight vascular congestion of the stomach and duodenum.

That the root of this plant contains a most energetic poison, and that it is capable of producing rapidly fatal effects, is proved by a case reported by Mr. Thomas, in which death took place in an hour. In May 1845, a child, aged five years, in good health, ate the bulbs of the Æthusa by mistake for young turnips. She was suddenly seized with pain in the abdomen, followed by sickness, but no vomiting. She complained of feeling very ill. On trying to eat, she could not swallow. She was incapable of answering questions, and her countenance bore a wild expression. The lower jaw became fixed, so as to prevent any thing being introduced into the mouth. She then became insensible, and died in *an hour* from the commencement of the symptoms: so far as could be ascertained, there were no convulsions. No post-mortem examination was made. A second child, aged three years, shortly after eating the same substance, was attacked with pain in the epigastrium, sickness, vomiting, and profuse perspiration. She soon recovered, with the exception of suffering severe griping pains without purging, but these disappeared the following day. A third child, of the same age, suffered from similar symptoms. Recovery in the two last cases was due to the plant having been eaten on a full stomach, and to the effect of early and copious vomiting. (Med. Times, Aug. 23, 1845, 408.) Mr. Thomas injected about two ounces of the juice expressed from the recent bulbs into the stomach of a dog through an aperture in the œsophagus, which he afterwards secured by a ligature. There were violent spasms and urgent attempts to vomit. In most of the animals upon which this experiment was tried, death took place in from one to four hours. The following case occurred to Mr. Stevenson. Two ladies partook of some salad, into which Æthusa cynapium had been put by mistake for parsley. They soon experienced a troublesome nausea, with occasional vomiting; oppressive headach, giddiness, and a strong propensity to sleep, at the same time that this was prevented by frequent startings and excessive agitations. There was a sensation of pungent heat in the mouth, throat, and stomach, with difficulty of swallowing, thirst, and loss of appetite. There was numbness, with tremors of the limbs. The two patients only slowly recovered from the effects of the poison. (Churchill's Botany.) These facts show that the Æthusa is a very poisonous plant.

ANALYSIS.—It is known from garden parsley by the smell of its leaves when rubbed, which is peculiar, disagreeable, and very different from that possessed by the leaves of parsley. The leaves of Fool's parsley are finer, more acute, decurrent, and of a darker green colour. Its flower-stem, which is striated or slightly grooved, is easily known from all other umbelliferous plants by the beard, or three long pendulous leaves of the involucrum under the flower. The flowers are white,—those of the garden parsley of a pale yellow colour.

The poisonous properties of this plant are believed to be due to an alkaloid, the chemical characters of which are unknown.

IPECACUANHA.

The powdered root of Ipecacuanha (CEPHAELIS IPECACUANHA) is well known to possess strongly emetic properties, and it is this which interferes with its action as a poison in ordinary doses. The properties of Ipecacuanha are dependent on the presence of an alkaloid, EMETINA, which forms about one per cent. of the powder or root. This is an uncrystalline powder of a light brown colour, but very soluble in water, alcohol and ether. It has a strong bitter taste. It is dissolved by nitric acid,—the solution acquiring a bright yellow colour. It is darkened by sulphuric acid, and on adding chromate of potash the liquid becomes red, and finally acquires a greenish-brown colour. Pelletier and Magendie found that from six to ten grains of the impure alkaloid given to animals caused violent vomiting, followed by stupor and death in about fifteen minutes. On inspection, the alimentary canal was observed to be inflamed. (Wibmer, op. cit. Cephaelis, 78.) Two grains of the pure alkaloid will kill a dog.

ANALYSIS.—For the chemical properties of Ipecacuanha powder see page 421, ante.

LABURNUM.

The bark and seeds of the common Laburnum (Cytisus Laburnum) contain an active poison called *Cytisine*. Dr. Traill met with two cases of poisoning by the seeds, and an interesting case, which was the subject of a trial at Inverness, has been more recently reported by Dr. Christison. (Ed. Med. and S. J. Oct. 1843.) A youth, with the intention of merely producing vomiting in one of his fellow-servants, a female, put some dry laburnum-bark into the broth which was being prepared for their dinner. The cook, who remarked a "strong peculiar taste" in the broth, soon became very ill, and in five minutes was attacked with violent vomiting. The account of the symptoms is imperfect; for the cause of them was not even suspected until six months afterwards. The vomiting continued thirty-six hours; was accompanied by shivering,—pain in the abdomen, especially in the stomach,—and great feebleness, with severe purging. These symptoms continued, more or less, for a period of eight months; and she fell off in flesh and strength. At this period she was seen by a physician, who had been called on by the law-authorities to investigate the case. She was then suffering from gastro-intestinal irritation, vomiting after food, pain in the abdomen, increased by pressure, diarrhœa, tenesmus, and bloody stools, with other serious symptoms. The medical opinion was that she was then in a highly dangerous state. The woman did not eventually recover until the following April. There was no doubt, from the investigation made by Dr. Ross and Dr. Christison, that her protracted illness was really due to the effects of the laburnum-bark.

Some experiments were then made on the action of the poison on animals. A teaspoonful of the powder of dry laburnum-bark was administered to a cat. Soon afterwards it writhed, apparently in great pain; in a short time it vomited violently, and, although languid and dejected for the rest of the day, it quickly recovered. Sixty-nine grains of the same powder were given to a dog. In ten minutes it whined and moaned, vomited violently, and soon got well. On a second occasion, twenty grains were found to act as a powerful emetic upon the animal. An ounce of the infusion of laburnum-bark, containing the active matter of sixty-two grains, was introduced by a catheter into the stomach of a full-grown rabbit. In ten minutes the animal looked quickly from one side to the other, twitched back its head twice or thrice, and instantly fell on its side in violent tetanic convulsions, with alternating emprosthotonos and opisthotonos, so energetic, that its body bounded with great force upon the side, up and down the room. Suddenly, however, all movement ceased, respiration was at an end, the whole of the muscles became quite flaccid, no sign of sensation could be elicited, and the animal died within *two minutes and a-half* after the poison was injected into the stomach. The body was opened in two minutes more, and the heart was found gorged with blood, but contracting with some force. The stomach was filled with green pulp, soaked with the infusion. No morbid appearance was visible any where. In repeating this experiment, one rabbit died in half an hour, another in three quarters of an hour, after small doses of the infusion were injected into the stomach; and a third rabbit speedily died after eating greens merely impregnated with the infusion. In all these instances convulsions were the leading symptoms produced. The same effects are popularly ascribed to the leaves, young pods, and seeds of the tree; but no experiments have been performed with these. The facts here detailed show that laburnum-bark is a most energetic poison—as powerful, even, as nux vomica.

Analysis.—There are no chemical means of detecting the nature of this poison, especially when administered in powder or infusion; or when a decoction of the bark is given in food. A decoction of the bark yielded a clear light brown infusion with a slight acid reaction. It was not precipitated by albumen, or a solution of tartarized antimony: hence it contained no tannic acid. With a persalt of iron it acquired a dark greenish-brown colour,—of a deep red by transmitted light. Strong nitric acid caused it to acquire a lighter colour. It gave a very copious gelatinous precipitate with acetate of lead, which was almost entirely redissolved by acetic acid. On decomposing this precipitate by sulphuric acid, filtering and applying a persalt of iron to the filtered liquid, a greenish-brown precipitate fell (gallate of iron) without any red tint whatever. A much stronger decoction of the bark, as well as a decoction of the tops, yielded similar results.

The bark has been said to contain meconic acid; but these results prove that none of this peculiar acid is present (p. 506, ante.) The only plan for determining with certainty the deleterious properties of the substance, would be by exhibiting a portion of the suspected decoction or infusion to animals.

LOBELIA.

The powdered leaves of Indian tobacco (LOBELIA INFLATA) contain an acrid principle which is capable of producing narcotico-irritant effects. As a poison it is but little known in this country. Wibmer relates that in one instance it produced at first violent vomiting in the person for whom it was prescribed; but the medicine was repeated until it was no longer ejected from the stomach. The patient suffered severe pain, and speedily died:—stupor and convulsions having preceded death. The powdered leaves and seeds are much employed by quacks in the United States; and accidents occasionally arise from the substance being prescribed in excessive doses. When administered in from ten to twenty grains lobelia acts as an emetic; but in larger quantity it acts deleteriously. It would also appear that even ordinary medicinal doses affect some individuals with great severity, owing, probably, to idiosyncrasy.

A case has recently occurred, in which a man lost his life by swallowing *one drachm* of the powdered leaves, prescribed by a quack. This person was seen by a medical practitioner, soon after he had taken the poison; he was evidently suffering great pain, but he was quite unconscious,—the pulse small, the pupils strongly contracted, and insensible to light. He had vomited the greater part of the poison. He suffered from spasmodic twitchings of the face, sank into a state of complete insensibility, and died in about thirty-six hours. On inspection, some fluid was found in the stomach, but none of the powder. The mucous membrane was intensely inflamed, and the vessels of the brain were strongly congested. (Pharm. Times, May 1, 1847, p. 182.)

ANALYSIS.—Lobelia is seen in the form of a greenish-coloured powder (fragments of leaves.) This powder acquires a reddish-brown colour from strong nitric acid, and is blackened by concentrated sulphuric acid. Iodine water has no effect upon it. The proto and persulphate of iron produce with it a dark green colour, the persulphate, very rapidly.

[Lobelia owes its properties to the presence of a peculiar alkaloid principle, called by Mr. W. Procter, who first obtained it pure, LOBELINA; its existence had previously been demonstrated by Dr. Calhoun.—G.]

MEADOW-SAFFRON.

Meadow-saffron (COLCHICUM AUTUMNALE) contains a poisonous alkaloid, the effects of which on animals are very analogous to those of veratria, the alkaloid existing in white hellebore. The most noxious parts of the plant are the bulbs (or roots) and seeds, but the leaves and flowers have also an irritant action. Toxicologists agree in placing this among the narcotico-irritant poisons, although in the cases yet reported there appear to have been no cerebral symptoms. Its effects are more like those of a vegetable irritant: it causes a burning pain in the œsophagus and stomach, with, in general, violent vomiting, and occasionally purging, followed by death. Dr. Ollivier has published the details of two cases, in which large doses of the tincture of colchicum root proved fatal. In each case about five ounces of the tincture were swallowed. In the first there was continued and violent vomiting, but no purging: the pupils were not dilated; pulse thready and slow; intense thirst; severe cramps in the soles of the feet; intellect unaffected; no convulsions or tetanic spasms. The patient died in twenty-two hours. The body was not inspected until putrefaction had advanced to a degree to destroy all the appearances. An unsuccessful attempt was made to extract colchicina from the contents of the stomach. In the second case symptoms speedily appeared, indicated by violent pain in the abdomen; frequent vomiting but no purging; difficult respiration; pupils not dilated; coldness of the surface; no tetanic spasms, but cramps in the soles of the feet; pulse small; intellectual faculties preserved. Death took place in twenty-eight hours. The vessels of the pia mater were much injected, but there was no injection or vascularity of the mucous membrane of the stomach. (Annales d'Hyg., 1836, ii. 394.)

A case is quoted in the Pharmaceutical Times, in which a man æt. 75, swallowed a large quantity of colchicum seeds. He soon experienced a burning sensation in the throat, with nausea, vomiting, violent colicky pains, and frequent diarrhœa. These were succeeded by difficulty of breathing, and discharge of bloody urine. After death, patches of inflammation and mortification were found in the stomach and duodenum. The latter contained some colchicum seeds. (Jan. 23, 1847, p. 354.) A softening of the mucous membrane of the stomach, has been observed in two cases of poisoning by tincture of colchicum.

In November 1839, a gentleman swallowed by mistake one ounce and a half of wine of colchicum. He was immediately seized with severe pain in the abdomen: other symptoms of irritation came on, and he died in seven hours. No post-mortem examination was required by the coroner! In another instance in which an ounce was taken, death occurred in thirty-nine hours. (Schneider's Annalen, i. 232.) In a well marked case of poisoning by the wine of colchicum, reported by Mr. Fereday, two ounces were taken. The symptoms did not come on for an hour and a half; there was then copious vomiting of a yellow fluid, severe pain with great tenderness in the abdomen, tenesmus, and thirst. The patient died in forty-eight hours without being convulsed or manifesting any sign of cerebral disturbance. The chief morbid appearance was a patch of redness in the mucous membrane of the stomach, near the cardiac orifice; the intestines were slightly inflamed. The head was not examined. (Medical Gazette, x. 161.) In another instance, where an ounce and a half of the tincture was taken, and death ensued in forty-eight hours, no morbid appearances were found. A man, aged fifty-two, took a decoction made with a table-spoonful of colchicum seeds to a pint and a half of water. He was seized with vomiting and purging, continuing incessantly until death, which took place in about thirty-six hours. The only appearance of note was that the stomach had a violet or purple hue. An interesting case of poisoning by the medicinal administration of colchicum has been communicated to me by Mr. Mann of Bartholomew Close. Three and a half drachms of the wine of colchicum were taken in divided doses, and caused death on the fourth day. There was no inflammation of the mucous membrane, but simply extravasation of blood into the mucous follicles.

The medicinal doses of the vinegar and wine of colchicum are from half a drachm to a drachm; of the tincture from twenty to thirty minims, and of the powder from two to eight grains. According to Dr. Aldridge, the tincture given frequently in medicinal doses, produces salivation. (Dub. Hosp. Gaz., Oct. 1845, p. 52.)

ANALYSIS.—Colchicum owes its noxious properties to the alkaloid COLCHICINA, which exists both in the seeds and root. It is in fine white crystals, which have a bitter acrid taste. It is soluble in water, has a feeble alkaline reaction, and forms crystallizable salts with acids.

MONKSHOOD.

Monkshood (ACONITUM NAPELLUS) which is also known under the names of *Wolfsbane* and *Blue-Rocket*, is a plant which grows freely in most parts of this country. The roots, seeds, and leaves are equally poisonous. They have a hot acrid taste, and when swallowed give rise to a burning sensation in the fauces, numbness and tingling in the limbs, swelling and pain in the abdomen, vomiting and diarrhœa, accompanied by vertigo, delirium, dimness of sight and other symptoms indicative of cerebral affection. In 1842, a lady residing at Lambeth was poisoned by her having eaten the *root* in mistake for horse-radish with some roast beef. It is not likely that, under these circumstances, much could have been taken; but very shortly after dinner slight vomiting came on, with severe pain in the abdomen. Emetics and the stomach-pump were used, but she died in three hours.

In the following case the *leaves* proved fatal. A boy, æt. 14, ate some of the leaves for parsley. In about two hours he complained of a burning sensation in the mouth, throat, and stomach, and vomited freely. Soon after this he fell on the ground in a fit; and seven hours after having taken the poison, he was found lying across the bed with his hands in his pockets, dead. On inspection, the cerebral vessels were filled enormously with dark-coloured fluid blood, upwards of a pound of which escaped from the skull and spinal canal. The stomach was empty: there was a deep inflammatory blush over the whole mucous surface, with patches of a darker colour. (Med.-Chir. Rev. July 1844, p. 261.)

An accident occurred at the hospital of Bordeaux, in which a dose of five grains of fresh *extract* of aconite was given to three patients. One of them died in three hours. In a quarter of an hour after taking the poison, the patients had tremors of the muscles, and a pricking or tingling sensation over their bodies; severe vomiting followed. They became quite unconscious; and on recovering their senses, there was confusion of sight, with intense headach; the skin was cold and clammy, the pulse slow and irregular, and the respiration short and hurried. Two of the patients recovered. (Med.-Chir. Rev., Oct. 1839, 544.)

A very complete medico-legal history of poisoning by aconite, has been published by Dr. Geoghegan in the Dublin Medical Journal. A trial for murder by poisoning with this plant took place at the Monaghan Lent Assizes in 1841, in which this gentleman was a witness for the crown. The medical evidence was beset with difficulties; for no trace of poison could be discovered, and it was only by a close analysis of symptoms and post-mortem appearances, that the charge was brought home to the prisoner. The deceased had eaten for his dinner some greens dressed for him by the prisoner; he complained of their having a sharp taste, and this was perceived also by another person present, who tasted them. It was ascertained that the deceased, soon after the meal, had vomited a greenish matter, and suffered from diarrhœa, restlessness, incoherence, trismus and clenching of the hands. He died in about three hours after having eaten the greens, but was not seen by a medical man while living. The chief appearance met with was in the stomach, where the mucous membrane was of a light reddish-brown colour. Traces of vegetable matter were found in the intestines: but no poison could be detected either botanically or chemically. The symptoms suffered by a friend of the deceased, who had accidentally tasted the greens, were very characteristic of poisoning by aconite. In *two* minutes he felt a burning heat in the mouth, throat, gullet and stomach; then a sensation of swelling in the face, with a general feeling of numbness and creeping of the skin. Restlessness, dimness of sight, and stupor almost amounting to insensibility, followed; and in about an hour after the meal, he was found speechless,—frothing at the nose and mouth, the hands and jaws clenched, appearing occasionally as if dead, and then again reviving. Vomiting, purging, tenderness of the epigastrium, cramps, tingling of the flesh, and a burning taste in the mouth, followed. He did not entirely recover until after the lapse of five weeks. The prisoner was convicted of murder, and confessed before execution that the powdered *root* of aconite had been mixed with pepper and sprinkled over the greens. From this case we learn that the actual discovery of a poison is not insisted on by a Court of law, when the medical and general evidence is conclusive of the fact of poisoning, (ante, p. 111.) Dr. Geoghegan, in the paper referred to, quotes two other instances of poisoning by aconite, one of a man aged fifty-six, who died in an hour and a quarter after eating the root; and the second, a boy aged seven, who died in two hours, having been much convulsed before death. One drachm of the root is said to have proved fatal; but it is probable that less than this would suffice to kill an adult. There appears to be considerable uncertainty in the operation of this poison under the form of *tincture*. In a case which occurred to M. Devay, (Cormack's Edinburgh Journal, April 1844,) a man recovered in three days after having taken upwards of ten drachms of the tincture, (only infused for a day,) while the late Dr. Male of Birmingham is reported to have died from the effects of not more than eighty drops taken in ten doses, over a period of four days,—the largest quantity taken at once being *ten* drops. (Prov. Med. and Surg. Journ., Aug. 20, 1845, p. 535, also, Med. Gaz. xxxvi. 861.) Dr. Pereira informs me that he has known general numbness produced in hysterical females by a dose of only *five minims* of a carefully prepared tincture. The preparations of aconite, even when used medicinally, are therefore likely to give rise to the most alarming symptoms, and even death. The common medicinal dose of the tincture is five drops three times a day; and of the powder or aqueous extract one or two grains.

The strength of this tincture is subject to great variation. The quantity of aconita contained in it is very uncertain: hence, in prescribing it, the intended strength should always be clearly defined. In the Pharmaceutical Journal for October 1847, no less than six different formulæ are given for its preparation, (p. 176.) The occasional weakness of the tincture may account for recovery after large doses, as in the following case. A patient swallowed about ten drachms of the tincture of Aconite (Parisian Codex.) The first symptoms were a sense of heat and constriction in the throat, great anxiety, restlessness, nausea, spasmodic contraction of the legs, coldness of skin, loss of vision, facies hippocratica, the head spasmodically drawn backwards, imperceptible pulse, stertorous respiration, insensibility of the skin, but perfect retention of intellect. After lasting about eight hours, pulsation returned and the symptoms abated. The patient recovered in three days. (Canstatt, Jahresbericht, 1844, v. 297.) Several cases of poisoning occurred some years since at Lille, in which tincture of the fresh roots of aconite was taken by mistake for a cordial. Three members of a family drank an ounce. The symptoms appeared in half an hour: there was severe burning pain in the throat and stomach, with vomiting, purging, tenderness of the abdomen, and delirium. One died in two hours; the second in two and a half hours; the third recovered. The only appearance met with on inspection, was great redness of the mucous membrane of the stomach and small intestines. (E. M. and S. J., xxviii. 452.)

A well-marked case of poisoning by a decoction of this plant occurred to Mr. Sayle. A man, æt. 39, boiled the fresh stalks and leaves of aconite in half a pint of beer until it was reduced to a quarter of a pint: he then swallowed half of it as a medicine. An hour afterwards he was found in bed, rolling his arms about and foaming at the mouth, pupils widely dilated, lower extremities paralyzed, surface cold and clammy, great nausea, pulse scarcely perceptible, and he was perfectly insensible. He soon afterwards died. The abdomen was examined, and the only appearance met with was a slight redness near the cardiac extremity of the stomach. (Med. Times, Oct. 18, 1845, p. 70.)

Dr. Fleming, who has closely investigated the subject of poisoning by aconite, considers that the poison may cause death—1, by producing a powerfully sedative impression on the nervous system; 2, by paralyzing the muscles of respiration, and causing asphyxia; and 3, by producing syncope. The last is the most common form of death in man, when the case is protracted for some hours. A dose sufficiently large to produce death in this way, excites, in the first place, numbness and burning heat in the mouth, throat, and stomach; pain in the abdomen, with sickness and vomiting; diminished sensibility of the surface; vertigo; dimness of vision, or complete blindness; ringing in the ears, and occasionally deafness; frothing at the mouth; sense of constriction in the throat, with sensations of weight and enlargements of various parts of the body, but especially of the face and ears; great muscular feebleness, with general trembling; more or less difficulty of breathing, and speechlessness; sinking at the pit of the stomach; pulse small, feeble, irregular, finally imperceptible; extremities and surface of the body cold and clammy; countenance blanched; and the lips bloodless. The individual dies suddenly;—the mental faculties are commonly retained to the last, or there is only slight delirium. The case generally proves fatal in from one to eight hours; if it last beyond this period, there is good hope of recovery. The most common appearance on inspection is general congestion of the venous system. The brain and membranes are gorged: in some instances there is increased vascularity of the mucous membrane of the alimentary canal. (An Inquiry on the Properties of the Aconitum Napellus, 1845, p. 43.) Some interesting cases of poisoning by the root of aconite are related by Dr. Pereira. (Mat. Med. ii. 1806.)

ANALYSIS.—The botanical characters of the plant may in some instances serve to identify it. Dr. Christison considers that its remarkable taste, which is at first bitter, but after a few minutes gives rise to numbness and tingling of the lips, will enable the analyst to distinguish it from other vegetable poisons. He recommends an alcoholic extract to be made of the contents of the stomach, and advises that this should be subjected to the sense of taste. Another mode of testing is suggested by the experiments of Dr. Pereira: when applied to the eye, the poisonous extract produces contraction of the pupil.

ACONITA.—The alkaloidal base of aconite, ACONITA, is the most formidable poison which has yet been discovered. It is contained in all parts of the plant, but especially in the root. It is a white or whitish-yellow semi-crystalline substance, somewhat resembling manna in appearance. It is soluble in alcohol and ether, but scarcely soluble in water, even on boiling; it is dissolved by acids (acetic) forming salts. It would be dangerous to attempt to taste this poison when pure, and all experiments with it should be performed with the greatest caution. So small a quantity as *one-fiftieth* part of a grain has endangered the life of an individual (Pereira, Mat. Med. ii. 1841;) and

Dr. Christison found that the tenth part of a grain introduced into the cellular tissue of a rabbit killed the animal in twelve minutes. It is even more powerful than pure prussic acid, and operates with tremendous rapidity. Dr. Roupell states, that the 1-100th of a grain will act locally, so as to produce a well-marked sensation in a part for a whole day. Its effects are similar to those occasioned by the plant. The medicinal preparations of aconite are apt to vary greatly in strength, in consequence of this alkaloid being sometimes strongly retained in the vegetable tissues. Nitric acid dissolves aconita without imparting to it any colour, in which respect this alkaloid resembles picrotoxia. Sulphuric acid produces with it no change of colour in the cold, but turns it of a dark-brown tint, when heat is applied. The solution of aconita in acetic acid is precipitated by potash, and the precipitate is not redissolved by an excess of the alkali. The specimen used in these experiments was absolutely pure: it was prepared by Mr. Morson.

There are other varieties of aconite, which are endowed with equally poisonous properties.

CHAPTER XLII.

NARCOTICO-IRRITANT POISONS CONTINUED—POISONOUS MUSHROOMS—SYMPTOMS AND APPEARANCES—DEADLY NIGHTSHADE—ATROPIA—NUX VOMICA—SYMPTOMS—APPEARANCES—STRYCHNIA AND ITS SALTS—ANALYSIS—BEAN OF SAINT IGNATIUS—UPAS AND WOURALI POISONS—OLEANDER—RUE—SQUILL—THORN-APPLE—SYMPTOMS—APPEARANCES—ANALYSIS—DATURIA—TOBACCO—ACUTE AND CHRONIC POISONING BY—NICOTINA—ANALYSIS—YEW-LEAVES AND BERRIES—INDIAN HEMP—SULPHATE OF QUININE—CARBAZOTIC ACID—NARCOTICO-IRRITANT VEGETABLES.

MUSHROOMS.

Poisoning by Mushrooms (Fungi) is by no means unusual as the result of accident. Modern writers on this subject have described no less than forty species, of which only a few can be safely eaten in this country. Among them the Agaricus campestris and esculentus are perhaps most commonly employed as articles of food. It is a curious fact, that the poisonous properties of mushrooms vary with climate, and probably with the season of the year at which they are gathered. Another circumstance deserving of notice, is, that by idiosyncrasy, some individuals are liable to be seriously affected even by those species which are commonly regarded as innocent. Some species which are poisonous in this country, are used freely by the Russians; although it appears they are in the habit of salting, boiling, and compressing them before they are eaten;—this may in some instances suffice to account for their having no noxious effects. Dr. Badham states that the *Agaricus campestris* or Common mushroom, which is largely eaten in England, is regarded as poisonous in Rome, and is accordingly rejected; while many varieties, which in this country would produce symptoms of poisoning, are eaten with impunity. There do not appear to be any satisfactory rules for distinguishing the mushrooms which are wholesome from those which are poisonous. The best test is that assigned by Dr. Christison—namely, that the poisonous vegetable has an astringent styptic taste; and perhaps also a disagreeable, but certainly a pungent odour. All mushrooms that are highly coloured, or grow in dark and shady places, are generally poisonous.

The noxious species of mushrooms act sometimes as narcotics, at others as irritants. It would appear from the reports of several cases, that when the narcotic symptoms are excited, they come on soon after the meal at which the mushrooms have been eaten, and that they are chiefly manifested by giddiness, dimness of sight, and debility. The person appears as if intoxicated, and there are singular illusions of sense. Spasms and convulsions have been occasionally witnessed among the symptoms where the case has proved fatal. Dr. Peddie has related three cases of poisoning by mushrooms (Edin. M. and S. J., xlix. 200,) in which the poison acted as a pure narcotic; there was no pain in the abdomen, nor irritation in the alimentary canal. The narcotic symptoms began in half an hour with giddiness and stupor; the first effect with one patient was, that every object appeared to him to be of a blue colour. The three patients recovered, two of them rapidly. When the drowsiness passes off, there is generally nausea and vomiting; but sometimes vomiting and diarrhœa precede the stupor. If the symptoms do not occur until many hours after the meal, they partake more of the characters of irritation;—indicated by pain and swelling of the abdomen, vomiting, and purging. Several cases, in which the symptoms did not appear until after the lapse of fourteen hours, are reported in the Medical Gazette (vol. xxv. p. 110.) In some instances the symptoms of poisoning have not commenced until after the lapse of thirty hours; and in these, narcotism followed the symptoms of irritation. It might be supposed that these variable effects were due to different properties in the mushrooms; but the same fungi have acted on members of the same family, in one case like irritants, and in another like narcotics. In most cases recovery takes place, especially if vomiting be early induced. In the few instances which have proved fatal, there has been more or less inflammation in the stomach and bowels, with turgescence of the vessels of the brain.

Balardini states, that of sixty-eight cases of poisoning by mushrooms, which occurred in the province of Brescia during a period of twenty years, twenty proved fatal. The principal symptoms were nausea, uneasiness in the abdomen, vertigo; a state resembling intoxication; vomiting and diarrhœa; loss of power of locomotion, with convulsions. (Canstatt's Jahresbericht, 1844, v. 300.) In six cases which occurred to Dr. Keber, in which the *Helvella esculenta* had caused symptoms of poisoning, the patients became jaundiced as soon as the vomiting had ceased. The principal symptom was urgent vomiting; but one girl, æt. 18, fell into a state of coma, from which she did not recover for three days. It was probable that, in this instance, the noxious effects were due to season. (Gaz. des Hôp., Oct. 10, 1846.) The common truffle (MORCHELLA ESCULENTA) has been known to give rise to severe symptoms of irritant poisoning. In some cases lately reported (Ed. Med. and Surg. Journ., Oct, 1845, 530,) it is probable that the truffles had undergone decomposition before they were eaten.

Ketchup, a liquor made from mushrooms, has occasioned faintness, nausea, and severe pain in the abdomen, disappearing only after some hours. (Dub. Med. Press, Sept. 24, 1845, 195.) There are two ways of explaining this effect: 1. either that the individual labours under an idiosyncrasy with respect to mushrooms in general; or 2. that noxious, have been gathered by mistake for esculent mushrooms. A case is on record which shows that a medical jurist may be easily misled when any active poison is mixed with and administered in a dish of mushrooms. A servant-girl poisoned her mistress, by mixing arsenic with mushrooms. This person died in twenty hours, after suffering severely from vomiting and colicky pains. On dissection, the stomach and intestines were found inflamed. Death was ascribed to the effects of the mushrooms, which were considered to have been unwholesome; and the fact of poisoning only came out many years afterwards, by the confession of the prisoner. This shows with what a watchful eye such cases should be examined: in the absence of poison from the stomach, it would be extremely difficult to develope the truth.

Analysis.—This discovery of portions of the fungus in the matter vomited, or the description of the food eaten, will commonly lead to a diagnosis of this form of poisoning. The poisonous principle contained in mushrooms is called *Fungin;* it appears to be of a volatile nature, and soluble in water; for some varieties of noxious mushrooms may be eaten with impunity, when they have been well boiled in water and afterwards pressed. One of the most poisonous in this country, *Amanita muscaria*, or Fly-mushroom, renders the water in which it is boiled so poisonous, that animals are killed by it, while the boiled fungus itself has no effect upon them. The liquid procured from it is used as a fly-poison, whence the name of the mushroom is derived. It is known by its rich orange-red colour.

NIGHTSHADE (DEADLY.)

There are several plants known under the name of Nightshade, which, however, differ much from each other. The Woody Nightshade (Solanum Dulcamara,) and the Garden Nightshade, or Solanum Nigrum, known by the red and black colour of their berries, have been elsewhere described. The vegetable poison now to be described is the Deadly Nightshade, or Atropa Belladonna.

Symptoms.—Within the last few years several well-marked cases of poisoning by belladonna have occurred, and in the autumn of 1846 many persons in this metropolis suffered from eating the ripe berries, which were openly sold in the streets as edible fruit! Two of these persons died, and the man who sold the berries was tried and convicted of manslaughter. The root and leaves are as poisonous as the berries; but the effects have been chiefly observed with the latter, which are not unfrequently eaten by mistake or accident. The symptoms produced by this poison are of a very uniform character, so that the description of one well-marked case might almost suffice for all the purposes of diagnosis. The following occurred at Guy's Hospital in August 1846. A boy, æt. 14, ate, soon after breakfast, about thirty of the berries, which he had bought in the street. In about three hours it appeared to him as if his face was swollen,—his throat became hot and dry,—vision impaired,—objects appeared double, and they seemed to revolve and run backwards. His hands and face were flushed, and his eyelids tumid: there were occasional flashes of light before his eyes. He tried to eat, but could not swallow on account of the state of his throat. In endeavouring to walk home he stumbled and staggered; and he felt giddy whenever he attempted to raise his head. His parents thought him intoxicated: he was incoherent,—frequently counted his money, and did not know the silver from the copper coin. His eyes had a fixed, brilliant, and dazzling gaze; he could neither hear nor speak plainly, and there was great thirst; he caught at imaginary objects in the air, and seemed to have lost all knowledge of distance. His fingers were in constant motion:—there was headach, but neither vomiting nor purging. He did not reach the hospital until some hours had elapsed; and the symptoms were then much the same as those above described. He attempted to get out of bed with a reeling, drunken motion: his speech was thick and indistinct. The pupils were so strongly dilated that there was merely a ring of iris, and the eyes were quite insensible to light. The eyelids did not close when the hand was passed suddenly before them. He had evidently lost the power of vision;

although he stared fixedly at objects as if he saw them. The nerves of common sensation were unaffected. When placed on his legs he could not stand. The pulse was 90, feeble, and compressible: his mouth was in constant motion, as if he were eating something. His bladder was full of urine on admission. He continued in this state for two days, being occasionally conscious; when, by a free evacuation of the bowels, some small seeds were passed: these were examined and identified as the seeds of belladonna. The boy gradually recovered, and left the hospital on the sixth day after his admission:—the progress of recovery was indicated by the state of the pupils, which had then only acquired their natural size and power of contraction. In three other cases which occurred at the same time, the berries having been baked in a pie, pains in the limbs, drowsiness, insensibility, and convulsions, were among the symptoms. In two cases of poisoning by the berries related by Dr. Moll, the symptoms bore a strong resemblance to those of delirium tremens, but among them were heat and dryness of the throat, loss of power of deglutition, incoherent speech, double vision, and strange spectral illusions, with occasional fits of wild and ungovernable laughter. On the following morning both of these patients recovered as if from a dream; but they suffered for some time from languor, thirst, and dryness of the throat: the pupils also continued dilated. (Casper's Wochenschrift, 10 Januar, 1846, p. 26.) Two very interesting cases of the effects of the berries on children are quoted in the Edinburgh Medical and Surgical Journal. (Vol. xxix. p. 452.) Among the first symptoms, three hours after the berries were eaten, the children were seized with uncontrollable fits of laughter; catching at objects; incessant, incoherent babble, and continual agitation of the body, with fixed staring eyes, and dilated insensible pupils. A man, æt. 34, ate about fifty berries to relieve his thirst. He immediately perceived a burning sensation in the throat, and a feeling of stupefaction. He staggered home and went to bed. In the evening he was seized with such violent delirium that it required three men to confine him. His face was livid; his eyes injected and protruding,—the pupils strongly dilated; the carotid arteries pulsating most violently; a full, hard, and frequent pulse, and loss of power to swallow. He was bled, and in about half an hour was able to swallow an emetic, which brought away a violet blue or purple liquid, (always a well-marked indication of this form of poisoning.) Purgative medicines and enemata were employed, and he recovered his consciousness in about twelve hours. (Case by Dr. Rosenberger, Canstatt's Jahresb, 1844, v. 295.) In six other cases, reported in the same journal by Dr. Teschenmacher, the symptoms varied slightly in the different patients. They all experienced double vision, dilatation of the pupils, constriction of the throat, vertigo, and a tendency to sleep. They who had eaten most berries fell into a soporose state, and had violent convulsions of the extremities. In twenty-four hours the whole of the family had recovered. (Ib. p. 296.)

The *root* of belladonna, administered in the form of decoction as a clyster, has destroyed life. Four scruples of the root were employed, and the liquid strained, and reduced to four ounces, was injected. After a slight stage of excitement, the patient, a female, æt. 27, fell into a state of complete coma; the countenance appeared swollen, and of a reddish-brown colour; the pupils were excessively dilated; the pulse was at first full and hard, then small: death took place in five hours. (Casper's Wochens. 8 Feb. 1845, p. 101.) This case proves that, in poisoning by nightshade, there is in some instances little or no delirium, and that the patient may be at once thrown into a fatal lethargy. A case of recovery, in which a girl, æt. 9, ate portions of the *bark* and masticated the *root* of belladonna, is reported by Mr. Bullock. (Med. Gaz. xix. 265.) In two hours there was sickness, lassitude, and dryness of the throat; in four hours delirium, with convulsions, came on; the face was distorted, eyes protruded, and pupils widely dilated. The girl was completely insensible. Under the use of the stomach-pump and emetics she recovered. (See APPENDIX.)

The *extract* of belladonna appears to be very uncertain in its operation. The medicinal dose is from one to five grains. In a case which occurred at St. George's Hospital, an ounce of the extract was taken without causing death; but in another instance, a child, æt. 9, nearly lost his life by a dose of thirty grains, administered to him in mistake for extract of taraxicum. Delirium came on in half an hour; this was followed by coma. In addition to other characteristic symptoms, the child suffered from convulsive twitchings of the arms. There was pain in the head with deranged vision, for ten days after the accident. (Prov. Med. Jour. Feb. 24, 1847, 98.) Dr. Gray, of New York, has related a case in which a child, between two and three years of age, swallowed from eight to twelve grains of the extract, and after suffering the usual symptoms in a severe form for three hours, recovered. This gentleman describes his own sensations after having taken a large dose of the same preparation. They bear out singularly the truth of the descriptions given by other observers. (See Med. Gaz. xxxvii. 255.) Orfila has satisfactorily accounted for these anomalies in the power of the extract. Some specimens are quite inert;—those only have an energetic action which are prepared by evaporating the fresh juice at a very low temperature. (Toxicologie, ii. 395.) The extract, as it is well known by its effects in dilating the pupil, acts through the skin. It is easily absorbed, and must therefore be used with caution. M. Casanova ordered a blister to be applied to the abdomen of a female, and prescribed a dressing of one part of extract of belladonna to three parts of mercurial ointment. At first, nine grains, and after two hours, thirty grains of the extract were thus employed. The patient was soon attacked with most violent delirium, crying out incoherently, and attempting to drive away horrible forms which she fancied she saw flitting around her. The pupil was enormously dilated; there was intense thirst with spasmodic constriction of the throat in drinking. These symptoms did not disappear until after the lapse of forty-eight hours. (Gaz. Méd. 13 Mars, 1847, 207.)

APPEARANCES.—The appearances observed in the cases which proved fatal in London during the autumn of 1846, were as follows: the vessels of the brain were congested with liquid blood; the stomach and intestines were pale and flaccid; there were some red spots towards the cardiac end. In other fatal cases, of which the post-mortem appearances have been reported, the vessels of the brain and its membranes have been found distended with thick black blood. Red spots have also been observed around the pharynx and œsophagus, and patches of a dark purple colour on the coats of the stomach. In some instances the mucous membrane has been completely dyed by the juice of the berries. The following case is of some interest. A boy, æt. 5, after having eaten a quantity of the berries of the belladonna, went to bed, was very restless, vomited once, and died in convulsions about fifteen hours after having taken the poison. On inspection, the eyes were half open, with an intense lustre; the pupils dilated; the mouth spasmodically closed, and the sphincter ani relaxed. The cerebral vessels were distended with dark coloured blood; the substance of the brain, cerebellum, and medulla oblongata, presented numerous bloody points. In the fauces and œsophagus there were several patches of redness. In the stomach there was some fluid, with three open berries; the mucous membrane was of a reddish-blue colour in various parts. (Case by Dr. Rosenberger, Canstatt's Jahresb. 1844, v. 295.)

ANALYSIS.—The indigestible nature of the husks and seeds will commonly lead to their detection in the matters vomited or passed by stool, or in the contents of the viscera after death. The seeds of belladonna are very small, and areolated on the surface; they cannot easily be distinguished from those of hyoscyamus. The colouring matter of the berry is of a deep-purple hue; it is turned green by alkalies, and red by acids. The leaves would be known by their botanical characters. Dr. Runge states that the urine, blood, or organic liquids containing this poison, applied to the eye of an animal, cause dilatation of the pupil. Orfila has not observed this effect in poisoning by belladonna (Op. cit. ii. 267,) and even if it occurred, he considers that it would be too vague a sign for diagnosis, as it takes place equally with hyoscyamus and stramonium.

Atropia.—The active principle of belladonna is an alkaloid, seen in white silky crystals. It requires 500 parts of water to dissolve it, but it is easily dissolved by alcohol, ether, or diluted acids. It has an alkaline reaction. A drop of its solution dilates the pupil of the eye; but this property is also possessed by hyoscyamia and daturia.

NUX VOMICA.

Nux vomica is the seed of the **Strychnos nux vomica**. Instances of poisoning by the powdered seed are by no means unfrequent. In 1837-8, there were three fatal cases marked in the coroner's return, and one case of poisoning by strychnia. The poisonous properties of nux vomica are due to the presence of strychnia; the symptoms in the two cases are alike, but of course much more severe when produced by the pure alkaloid. Nux vomica is usually taken in the form of powder. It is retailed to the public by druggists at the price of eightpence an ounce.

Symptoms.—The powder has an intensely bitter taste, which is very persistent. In from five to twenty minutes after it has been swallowed, the patient is suddenly seized with tetanic symptoms, affecting the whole of the muscular system, the body becoming rigid, the limbs stretched out, and the jaws so fixed, that considerable difficulty is experienced in introducing any thing into the mouth. The muscles of the chest are also fixed by spasmodic contraction, and the body sometimes assumes the state of opisthotonos: the intellect is clear. The spasmodic state ceases, but after a short interval reappears; and the chest is so fixed, as to give the feeling of impending suffocation. After several such attacks, increasing in severity, the patient dies asphyxiated. Drowsiness and a feeling of general illness have sometimes preceded the attack; vomiting, pain in the abdomen, and other symptoms of irritation, have been occasionally witnessed where the case was protracted; but, in general, death takes place long before such symptoms are manifested.

Post-mortem appearances.—In a well marked case of poisoning by this substance at University College Hospital, in 1839, the only appearances met with, were general turgescence of the brain and its vessels. A quantity of the powder was found in the stomach, to the mucous membrane of which it adhered very tenaciously; there was injection, with many ecchymosed points at the cardiac extremity. The brain, as well as the spinal marrow, has been found softened. The spasmodic condition of the body has been observed to continue after death, and to pass into the state of cadaverous rigidity.

Quantity required to destroy life—Period of Death.—The smallest fatal dose yet recorded, is three grains of the alcoholic extract of nux vomica; but it is not stated to how much of the powder this would correspond. Two cases occurred in London, in 1839, in each of which fifty grains of the powder (equal to one-fourth of a grain of strychnia) proved fatal. In one of these death took place in an hour; the chemist who sold the poison said that he did not think a dose of fifty grains was sufficient to cause death; but a much smaller quantity has been known to destroy life. One case proved fatal, where thirty grains of the powder were taken in two doses; (Christison, 901;) and in another reported by Dr. Traill, *fifteen grains* destroyed life; this is probably *the smallest* fatal dose of the powder yet known. (Outlines, 137.) Death usually occurs in from one to two hours; but Dr. Christison quotes a case where a man died in *fifteen minutes* after taking a dose. (898.) This is probably the shortest period. There are several instances of recovery on record. Sobernheim mentions the case of a young man, who took half an ounce of the powder, and suffered from the usual symptoms: emetics were administered, and he recovered. A second occurred to Dr. Basedow, of Merseburg. A young lady swallowed, by mistake, a table-spoonful of the powder; she was almost instantly deprived of the power of walking, and fell down, but did not lose her recollection. There was great difficulty of breathing. Emetics were administered with good effect, and she recovered. A third case is described by Mr. Baynham, of Birmingham. A girl, aged twenty, swallowed half an ounce of the powder. In half an hour the usual tetanic symptoms came on, but she was perfectly sensible. In administering remedies, the spasm of the muscles of the jaw was such, as to cause her to bite through the cup. The convulsions gradually subsided in about four hours from the first attack; and the next day, although feeble and exhausted, she was able to walk home. (Med. Gaz. iii. 445.) The reporter of this case states that he has often prescribed a scruple of powdered nux vomica daily, without any injurious effects following! For another case of recovery, in which half an ounce was taken, see Prov. Med. Journal, Jan. 7, 1846, page 5.

TREATMENT.—The removal of the poison from the stomach by emetics, or the use of the stomach-pump, must be chiefly relied on. Unless these means be employed early, the jaw may become spasmodically fixed, so as to render all efforts at relief unavailing. In general, however, the spasms have intermissions, so that there may be time to apply remedies in the interval.

STRYCHNIA AND ITS SALTS.

SYMPTOMS AND APPEARANCES.—The *symptoms* produced by strychnia closely resemble those described in speaking of nux vomica. The following case is reported in the Lancet, Jan. 7, 1838. A young man, aged seventeen, swallowed forty grains of strychnia. The symptoms came on in about a quarter of an hour; trismus and spasmodic contraction of all the muscles speedily set in; the whole body becoming as stiff as a board; the lower extremities were extended and stiff, and the soles of the feet concave. The skin became livid, the eyeballs prominent, and the pupils dilated and insensible; the patient lay for a few minutes without consciousness, and in a state of universal tetanus. A remission occurred, but the symptoms became aggravated, and the patient died asphyxiated, from the spasm of the chest, in about an hour and a half after taking the poison. *Appearances:* On inspection, twenty hours after death, the body was very rigid. There was effusion in the spinal sheath, and the upper part of the spinal marrow was softened; the brain was congested, but the alimentary canal was in its normal state. In a case by Dr. Edward, in which about twelve grains had caused the death of a young man, the body was strongly rigid, and the left foot concave by contraction. There was turgescence of the vessels of the brain; the heart distended, and containing dark fluid blood. The other organs were healthy. (Med. Gaz. xxxvii. 833.) Similar appearances were met with in another case, reported by Mr. Evans in the same journal, except that the heart contained coagulated blood. (Ib. p. 925.) In no instance has there been any appreciable morbid change in the alimentary canal.

Diagnosis.—The symptoms produced by strychnia very much resemble those of tetanus: but in the last-mentioned disease they are more slowly formed, and can only be coincidentally connected with the taking of some kind of solid or liquid. Death is a much more rapid effect of the poison, than of the disease as it is produced by natural causes. Medical men may, however, be easily deceived respecting the origin of the symptoms, when the dose is small and frequently repeated. A few years since, an action was brought against an Insurance Company, to recover the amount due on a policy for the life of a young lady. She died under very suspicious circumstances, after several insurances on her life had been effected by the plaintiff in the cause. The party did not recover in the action, and he ultimately fled the country. It was rendered probable afterwards, that he had destroyed the deceased by administering to her strychnia in porter.

QUANTITY REQUIRED TO DESTROY LIFE.—Three-eighths of a grain of strychnia, given medicinally, gave rise to violent tetanic convulsions, spasms of the extremities, trismus, opisthotonos, spasmodic fixing of the chest, and all the other formidable symptoms usually produced by this alkaloid. (Edin. Med. and Surg. Journal, xlix. 327.) Half a grain, given three times a day for several days, caused death under the usual symptoms. (Pereira, ii. 1310.) Thus we see that this substance, which is almost insoluble, requiring seven thousand parts of water to dissolve it, is capable of exerting a powerfully poisonous action. I have been informed of a case, where a person took at a dose a *grain* of strychnia; vomiting supervened, and in the course of a few hours the patient recovered. When medicinally employed, and the dose is gradually raised, the system may become habituated to the poison, so as to resist the effects of very large doses. In a case in which the dose was gradually increased, Dr. Pereira has seen one grain and a half taken without causing injurious effects; and even three grains have been taken daily for some time by a patient, without dangerous consequences. (Gaz. Méd. Mars 1845.) The ordinary medicinal dose of strychnia or its salts, is from the sixteenth to the eighth of a grain.

That a very small quantity of strychnia is sufficient to destroy life, may be inferred from its fatal effects in several cases of recent occurrence. A young woman was lately killed by a dose of *three grains*, prescribed by mistake for nux vomica. She died in two hours under the usual symptoms. (Med. Gaz. xxxvii. 254; and Prov. Med. Journ. Dec. 24, 1845.) In Sept. 1845, a case occurred to Dr. Watson, in which a girl, æt. 13, swallowed three pills, containing each a quarter of a grain of strychnia. In twenty minutes she became convulsed, the face flushed, lips livid, and the whole of the muscles of the body rigid. There were spasms of the diaphragm; and a fit of convulsions took place at intervals of a few minutes: the mind was quite clear. The girl died in an hour; and the only post-mortem appearance was congestion of the lungs. In this instance the fatal dose was *three-quarters of a grain*, and the effects were violent and well marked. (Med. Gaz. xxxvii. 253.) The *smallest dose* which has yet been known to prove fatal, was in the case of Dr. Warner, who swallowed *half a grain* of the sulphate of strychnia by mistake for morphia. In a few minutes he was seized with constriction of the throat, tightness of the chest, rigidity of the whole muscular system, and tetanic convulsions. He died in about *fourteen minutes*, *i. e.* within the shortest period of time yet known, from the effects of this poison. (Brit. Amer. Journ. Aug. 1847, 106; and Med. Gaz. xl. 477.) In one case, in which twenty grains were taken dissolved in alcohol, the symptoms did not commence for nearly a quarter of an hour, and the individual died in half a hour. (Casper's Wochenschrift, 28 February, 1846, 143.)

ANALYSIS.—Nux vomica is well known as a flat round kernel, less than an inch in diameter, with radiating fibres, slightly raised in the centre. It is of a light brown colour, and covered with a fine silky down. It is very hard, brittle, tough, and difficult to pulverize. The powder is of a grey brown colour, like that of liquorice: it is sometimes met with in a coarsely rasped state:—it has an intensely bitter taste. It yields to water and alcohol strychnia, brucia, igasuric or strychnic acid, and some common vegetable principles. Heated on platina-foil, it burns with a smoky flame. Nitric acid turns it of a deep orange-red colour, which is destroyed by protochloride of tin. The aqueous infusion is similarly changed by nitric acid, and it is freely precipitated by tincture of galls. The quantity of strychnia contained in the powder has not been very accurately determined. It probably amounts to about 0.5 or one-half grain per cent. If this be the case, the strychnia is more energetic when contained in the nut, than when separated.

If nux vomica has been taken in the form of powder, we can only identify it in the stomach by demonstrating the presence of its strychnia. As the powder is quite insoluble in water, it may generally be separated by decantation.

Organic liquids.—Various processes have been suggested for the separation of strychnia from nux vomica; but, owing to the very small quantity of the poisonous alkaloid contained in it, it is obvious that, unless we have a large quantity of the powder to examine, none of these are likely to succeed. Fifty grains of the powder will not yield more than one quarter of a grain of strychnia. The following is, perhaps, the most simple process: Digest the powder in alcohol of about seventy per cent., until nothing further is dissolved. Evaporate to an extract, and boil this in water with a small quantity of calcined magnesia. Strychnia, mixed with brucia, is thereby precipitated: and may be separated from the magnesia in the insoluble residue, by further digestion in boiling alcohol. This alcoholic liquid yields strychnia, which may be purified in the usual way. There are no chemical characters by which the acid, united to the strychnia, can be readily identified; and thus this process is more defective than that for morphia, since we acquire so much more certainty, where, besides the poisonous base, we can show by tests the presence of the peculiar acid with which the base is known to be united. Another method of separating strychnia, is by making an aqueous infusion with very diluted sulphuric acid, and afterwards precipitating the strychnia by boiling the filtered liquid with lime. The aqueous infusion of nux vomica gives the same bright red tint with nitric acid, as the infusion of opium; but it is known from the latter by its giving a green instead of a deep red colour with the permuriate of iron; as well as by the bleaching action of chloride of tin. Infusion of galls precipitates it abundantly.

Tests for strychnia.—Strychnia is known by the following properties: 1. It may be met with, crystallized in short prisms or in the state of a greyish white powder of an intensely bitter taste. 2. It is scarcely soluble in water, hot or cold;—it is not very soluble in alcohol, but is dissolved by ether and acetic acid. 3. When heated on platina-foil, it melts, becomes charred, and burns with a black smoky flame, leaving a residue of carbon. This experiment should be performed with caution, and on the smallest quantity:—if the vapour be respired, it might give rise to alarming symptoms. 4. When the crystals are dropped into strong nitric acid, they become slowly dissolved, without evolving deutoxide of nitrogen, and the liquid acquires a red colour. This colour is immediately destroyed by chloride of tin; but if allowed to remain exposed to air, it slowly acquires a dark greenish brown hue. This red colour, thus given by nitric acid, appears to be caused by the presence of brucia. I have, however, found it to be more or less produced in all the specimens of uncombined strychnia which I have tried, and it is pretty certain, that no specimen of strychnia is ever likely to come before a medical jurist in practice, which does not possess the property of being turned red by nitric acid, although *pure* strychnia and its salts are certainly not thus affected. 5. If to strychnia in water a few drops of diluted acid (sulphuric) be added, it is readily dissolved on boiling,—crystals being sometimes deposited on cooling. 6. This solution is precipitated by tincture of galls. 7. It is precipitated white by alkalies (ammonia). 8. It is precipitated yellow-white by chloride of gold, while a salt of morphia gives with this test on boiling a precipitate of reduced gold. (See 13, infra.) 9. It is coloured red by nitric acid;—the colour being discharged by chloride of tin. 10. It is not affected by permuriate of iron, or by iodic acid and starch—characters whereby it is easily known from morphia. 11. Potash precipitates a salt of strychnia readily, and the precipitate is not easily redissolved by the alkali: it only precipitates a salt of morphia when much concentrated, and the precipitate is immediately dissolved by a slight excess. 12. Independently of these characters, the action of sulphuric acid and chromate of potash on strychnia and many other alkaloids, is very different. Strong sulphuric acid produces no change of colour in pure strychnia; but if a drop of a solution of chromate of potash be added, the mixture speedily acquires a crimson, passing to a deep blood-red colour. If to this mixture of sulphuric acid and strychnia, a grain of peroxide of lead, or of peroxide of manganese be added, the most splendid colours are brought out, passing rapidly through all the shades of blue, violet, purple, and crimson, into a blood-red colour. This singular property, first pointed out by Marchand, Mack, and Otto, so far as I have been able to ascertain, is possessed only by strychnia. 13. Strychnia dissolved in acetic acid gives a dense yellowish-white precipitate with chloride of gold. This is not redissolved on boiling, nor is the gold reduced; but the liquid acquires a pink-red colour. Morphia, under the same circumstances, gives no precipitate with chloride of gold; and metallic gold is immediately set free on boiling the mixture.

BRUCIA.—The seed of the nux vomica tree yields chiefly strychnia, with a small proportion of another poisonous alkaloid called BRUCIA; while the bark of the tree yields brucia in large, and strychnia in small proportion. This bark was for a long time supposed to be the Angustura, or Cusparia, which is used in medicine; and it is now sometimes called the FALSE ANGUSTURA. Before this serious mistake was discovered, several fatal accidents occurred. The nux vomica bark is just as poisonous as nux vomica itself. The alkaloid brucia acts on the body like strychnia, but it is much less powerful. It is considered to have only about one-twelfth of the strength of strychnia.

ANALYSIS.—*Pure* brucia is a white crystalline solid. 1. It is soluble in boiling water, but precipitated on cooling; its hot solution has a strong alkaline reaction. 2. It is easily dissolved by alcohol or acetic acid. 3. It is decomposed by heat, and burns in the air. 4. Strong nitric acid readily dissolves it, forming a brilliant orange-red coloured solution; this colour is very persistent. 5. Strong sulphuric acid strikes with it a rich pink colour, which is deepened on the addition of a drop of a solution of chromate of potash, passing to an orange-red, and finally a greenish-brown colour. 6. Peroxide of lead and peroxide of manganese do not produce with it, when mixed with sulphuric acid, the brilliant colours observed in the case of strychnia. 7. Potash precipitates brucia from its solution in acetic acid, but redissolves the precipitate much more readily than in the case of strychnia.

It will be seen from this description, that there are some striking differences between brucia and strychnia. The results here described were obtained from very pure specimens, prepared by M. Morson. It is rare, however, to meet with brucia unmixed with strychnia, and *vice versâ*: hence the analyst will not be surprised to find that the alkaloids commonly sold as distinct, do not differ from each other in their chemical properties.

Bean of Saint Ignatius.—This is the seed of the **Strychnos Ignatia.** In the powdered state it is an active poison, and produces effects like those caused by nux vomica. This bean is said to contain from one to two per cent. of strychnia.

Analogous to the bean of Saint Ignatius is the fruit or kernel of the *Tanghinia Venenifera*, which grows in Madagascar. It is stated to be a poison of extraordinary energy; but very little is known concerning it. It contains an uncrystallizable substance, *Tanghin*, upon which its poisonous properties depend.

Upas Tieute.—This is the aqueous extract of the bark of a large climbing shrub, **Strychnos Tieute**, which grows in Java. It contains strychnia, but no brucia. Its action is identical with that of the former alkaloid. This poison must not be confounded with the Upas poison of Java, which, according to Pereira, is derived from a large forest tree (Antiaris toxicoria,) growing to the height of from 60 to 100 feet. The milky juice contains 3·56 per cent. of a poisonous principle called Antiarin. It acts upon the brain and spinal marrow, and, according to Sir B. Brodie, paralyses the heart.

Wourali poison.—This, which is a South American poison, is known under the various names of woorara, oorary, urari, curara or ticunas, according to the district in which it is made. It is the extract of the strychnos toxifera or guianensis. The effects of this poison have been much exaggerated. Animals have been said to fall instantly dead when shot with an arrow poisoned by Wourali; but this has only been in cases where a vital organ like the heart has been wounded, and then death was not due to the poison. According to Mr. Waterton, the poisonous extract is procured chiefly from the bark of a creeper or vine called wourali (strychnos guianensis,) which grows in the forests of Guiana and Central America. The Indians prepare the poison with a great deal of mystery, and mix with it other herbs, red and black ants, and the pounded fangs of a venomous snake. The juice is extracted from the stem of the creeper by infusion and compression; it is then heated with the other ingredients over a slow fire until it acquires a dark colour. It is afterwards put into a small pot carefully covered over and kept in a dry place. It is occasionally warmed over a fire to prevent the effects of dampness.

The extract is so exceedingly miscible with water, that the slightest moisture dissolves it; hence, it speedily diffuses itself when introduced into a wound. The symptoms which it produces in animals are stupor, paralysis and convulsions. It does not begin to produce any apparent effects until after the lapse of one or two minutes, and there is apparently no pain: convulsions come on in two or three minutes, and the animal dies in four or five minutes. Putrefaction is not accelerated, and the flesh of the game thus killed is used as food without any serious effects resulting. This is probably due to the very small quantity of poison present; for the Wourali, in a sufficient dose, is fatal to all animals; and there is no reason to believe that it is decomposed in the act of absorption. It requires much more of this extract to kill an ox than a smaller animal; thus, the Indian adjusts the size of his arrow and the quantity of poison to the size of the animal. In one experiment, three arrows were introduced beneath the skin of an ox. For four minutes there was no effect: the animal then set itself firmly on its four legs as if to resist falling, and remained quite still for fourteen minutes. It then attempted to walk, staggered, and fell. The eyes became fixed, dim, and apparently insensible to light. Convulsions appeared in the legs; there was emprosthotonos, laborious respiration, and an escape of a frothy liquid from the mouth. The convulsions in the extremities gradually ceased: there was still a perceptible action of the suffering, however, from indistinctness of vision and vertigo. (Med. Gaz. viii. 605.) The seeds of this plant have been known to produce furious delirum; and a case is mentioned by Sauvages, of an old man of sixty, who, after taking the poison, became intoxicated, maniacal, and lost the power of speech. He remained in a lethargic state for five hours. Several fatal cases are reported, one of which terminated in six hours. Dr. Thomson relates the case of a child, aged two years, who swallowed sixteen grains of the seeds. Maniacal delirium supervened; the symptoms resembled those of hydrophobia, and death took place in twenty-four hours. A case which occurred to Dr. Schlesier ended more fortunately. A boy, æt. 4, mistaking the fruit of the thornapple for the heads of poppies, ate a quantity of them. Dr. Schlesier saw him soon afterwards: his face was flushed, his eyes glistening and in constant motion; the pupils exceedingly dilated, and the countenance was that of an intoxicated person. He sat up in bed quite unconscious, but continually babbling and occasionally starting up suddenly; his hands apparently directed at imaginary objects in the air. His pulse was very slow: there was no fever, but intense thirst and violent perspiration from incessant motion. Emetics and enemata were administered, which had the effect of bringing away a large quantity of stramonium-seeds; the boy fell into a sound sleep, and recovered the following day. (Canstatt's Jahresb., 1844, v. 297.) In the Lancet (April 26, 1845, p. 471,) a case is quoted from the Boston Journal, in which three females swallowed an infusion of stramonium-leaves for horehound. They were found lying in bed, stupid, unable to articulate, with a peculiar wildness of countenance, and flushed face; the pupils dilated and insensible; conjunctivæ highly injected; lips and tongue parched; no vomiting, breathing at times stertorous and laboured; hands cold, with a trembling and slightly convulsive movement, great rigidity of the muscles of the neck and back, and occasionally active efforts at utterance. Stimulants were administered with benefit in two cases; the third proved fatal. Mr. Sobo met with the case of a child, æt. 5, who ate more than a drachm of the seeds slightly roasted. In about an hour he appeared much excited and delirious, pulse 120, face flushed, eyes of a dazzling lustre, and pupils dilated; there were convulsive motions of the limbs and neck, with thick frothy saliva about the mouth. Emetics were given; some stramonium-seeds were ejected, and more were brought away in the evacuations by a full dose of castor oil. In three days the boy had perfectly recovered. (Med. Times, Oct. 9, 1847, 650.) Paralysis and delirium have been witnessed among the symptoms, which on the whole bear a very remarkable resemblance to those occasioned by belladonna. The detection of the seeds in the vomited matters or in the fæces, will be a certain means of diagnosis.

In a singular case which became the subject of a trial at Osnabrück, a woman administered to her mother, a decoction of the bruised seeds of the thornapple, of which it is supposed, there were about 125. She very soon became delirious, threw her arms about, and spoke incoherently; she died in seven hours. (Henke, Zeitschrift der S. A. 1837, i. H.) The seeds retain their properties notwithstanding exposure to heat: thus the smoking of stramonium-seeds is attended with danger. One of the methods of poisoning adopted by the Hindoos, not so much with the intention of destroying life as of facilitating the perpetration of robbery, consists in administering to persons a strong decoction of the seeds in curry, or some other highly flavoured article of food. Delirium and insensibility soon follow, and sometimes death is the result; but no suspicion of the real cause appears to be excited. The local application of the bruised leaves, seeds or fruit, to an abraded portion of skin, may give rise to all the effects of poisoning. Death may take place even although the whole of the seeds have been ejected, provided they have remained in the body for a sufficiently long period to allow of the absorption of the poisonous principle. This happened in a case reported by Mr. Duffin,—that of his own child, æt. 2, who swallowed about one hundred seeds of stramonium, weighing sixteen grains: the usual symptoms were manifested in an hour, and the child died in twenty-four hours, although twenty seeds had been ejected by vomiting and eighty by purging. (Med. Gaz. vol. xv. 194.)

The *Extract* of stramonium possesses the properties of the seeds, producing, in an over-dose, dryness of the fauces, intoxication, and delirium. Dr. Traill has known two cases of poisoning by this substance, in one of which eighteen grains were taken by mistake for extract of sarsaparilla. (Outlines, 141.) The medicinal dose of the extract is about a quarter of a grain; of the powdered seeds, half a grain; and of the leaves, one grain.

APPEARANCES.—In a well-marked case of poisoning by stramonium-seeds, in which death took place in less than eight hours, Mr. Allan found the following appearances:—great turgescence of the vessels of the brain and its membranes,—the brain firm and highly injected,—choroid plexus turgid,—ventricles containing serum,—substance of the lungs vascular,—heart flaccid: the stomach contained about four ounces of ingesta, consisting of food mixed with eighty-nine seeds of stramonium. There were two patches of extravasation in the mucous coat: one on the larger curvature, and the other near the pylorus,—many seeds and fragments were also found in the intestines. (Lancet, Sept. 18, 1847, p. 298.) In the Osnabrück case, there were marks of diffused inflammation about the cardia. In Mr. Duffin's case, there was nothing remarkable in the condition of the brain or its membranes:—no seeds were found in the intestinal canal.

[Owing to the frequent occurrence of this plant in the United States, numerous cases of poisoning by it have been reported. In many of them, in addition to the usual symptoms of narcotic poisoning, an eruption appeared on the skin.—G.]

ANALYSIS.—The *seeds* of stramonium, from which accidents have most frequently occurred, are flattened, kidney-shaped, rough, and of a dark-brown or black colour.

DATURIA.—The poisonous properties of this plant are owing to the presence of the alkaloid DATURIA, which forms about one per cent. of the dried vegetable. This substance crystallizes in colourless quadrangular prisms: it has a bitter taste, becoming acrid, and resembling that of tobacco. It is highly poisonous: the eighth of a grain killed a sparrow in three hours. When dissolved and placed on the eye of an animal, it causes excessive dilatation of the pupil, which lasts for some days. It is readily dissolved by boiling water, and the solution has a strong alkaline reaction. In its chemical properties it closely resembles hyoscyamia and atropia.

The absorption of this poisonous alkaloid is doubtless the cause of the symptoms. Mr. Allan, in the case just related, states that he obtained from six ounces of urine, taken from the bladder of the deceased, crystals of daturia; but they appear to have been of an entirely different form, *i. e.* pentahedral or polyhedral plates, instead of quadrangular prisms. They resembled daturia only in causing dilatation of the pupil when dissolved in water and the solution was dropped into the eye. Their form appears to have been that assigned to cystin by microscopical observers. (Bird's Urinary Deposits, 146.)

TOBACCO.

The leaves of the NICOTIANA TABACUM, variously prepared, either as tobacco or snuff, exert a very powerful action on the system, although fatal cases of poisoning by tobacco are by no means common. The effects which this substance produces, when taken in a large dose, either in the form of powder or infusion, are well-marked. The symptoms are nausea, vomiting, vertigo, delirium, loss of power in the limbs, general relaxation of the muscular system, trembling, complete prostration of strength, coldness of the surface with clammy perspiration, convulsions, paralysis, and death. In some cases there is diarrhœa, with violent pain in the abdomen; in others there is rather a sense of sinking or depression in the cardiac region, passing into syncope, or creating a sense of impending dissolution. With the above-mentioned symptoms there is dimness of sight, with confusion of ideas,—a small, weak, and scarcely perceptible pulse, and difficult respiration. Dr. M'Gregor has seen some of the most severe symptoms follow the administration of an enema which contained only half a drachm of tobacco in the form of decoction. (Lancet, Aug. 30, 1845, 240.) Dr. Paris witnessed a case, which proved rapidly fatal, where a decoction of tobacco had been used as an enema in the attempted reduction of strangulated hernia (Med. Jur. ii. 418;) and several cases of a similar kind are recorded by other writers. Dr. Periera considers that it would not be safe to use more than fifteen or twenty grains under these circumstances, and he quotes an instance from Dr. Copland, in which death was caused by so small a dose as *thirty grains.* (Mat. Med. ii. 1254.) The fatal effects of tobacco may follow very speedily on its administration. Death has been known to take place in so short a period as three-quarters of an hour; and a case which occured to M. Tavignot is reported to have terminated fatally in *eighteen minutes.* (Brit. and For. Med. Rev. No. xxiv. 562.)

According to M. Demarquay, there is a peculiarity in the action of tobacco, in poisonous doses, on animal heat. There are some substances, such as morphia, which tend to lower the temperature of the body; others such as digitalis, belladonna, and strychnia, increase it. Tobacco neither raises nor depresses it. (L'Union Médicale, Juillet 24, 1847, 371.) This statement is not borne out by observation, as under the influence of tobacco there is often great coldness of the surface.

Tobacco acts *locally* as a poison: thus, when applied to wounded, abraded, or diseased surfaces, in the form of powder, juice, or as a decoction of the leaves, it may occasion the most alarming symptoms, and even death. (Orfila, ii. 404.) This fact is of importance, as some quack-remedies for skin-diseases are formed of tobacco.

There have been but few instances in which the bodies of persons, poisoned by tobacco, have been inspected. Orfila found, on examining the body of a dog killed by this substance, that the mucous membrane of the stomach was strongly reddened throughout. From the insolubility of the fibrous portion of tobacco, it is most probable that some of it would be found in the stomach on inspection.

Tobacco is rarely administered medicinally in substance. In a dose of five or six grains, *Snuff* acts as a powerful emetic, and in larger doses it produces symptoms of poisoning. It is a remarkable instance of the effect of habit, that the quantity thrust into the nostrils, as a sort of morbid luxury, does not appear to produce any directly noxious effects on the system. The diurnal allowance of many snuff-takers, introduced into the rectum in powder, would most probably give rise to serious symptoms, in one whose system was not habituated to the use of tobacco. The same may be observed of chewing tobacco, and *smoking:* in the latter case, the volatile oil of tobacco is brought into immediate contact with the mucous membrane, producing vertigo and sickness, in those not accustomed to the practicé. In two instances, in which a large quantity of tobacco was consumed by smoking, death was the result. This involves a question as to the *chronic* form of poisoning by tobacco. Drs. Prout, Laycock, and Wright, consider that habitual smoking is injurious to health, because it is liable to disorder the digestive functions. This is denied by others, on account of the difficulty of showing that the health of inveterate smokers is damaged by the habit, or that their lives are shortened by it. Dr. Prout's view appears to me, notwithstanding, quite reasonable. A poisonous substance like tobacco, whether in powder, juice, or vapour, cannot be brought in contact with an absorbing surface like mucous membrane, without in many cases producing disorder of the system, which the consumer is probably quite ready to attribute to any other cause than that which would render it necessary for him to deprive himself of what he considers not merely a luxury, but an article actually necessary to his existence. The argument that cases cannot be adduced to show direct injury to health, proves too much, for the same difficulty exists in respect to the habit of opium-eating. (p. 474, ante.) On this subject see Med. Gaz. xxxviii. 590, and Lancet, Aug. 1845, 240. Some doubt has existed whether the vapour of this substance, in tobacco manufactories, is or is not injurious to the health of the workmen employed. M. Parent-Duchâtelet considered that after a time, it had no influence on health. The more recent researches of M. Melier have, however, led to the conclusion, that the vapours long respired are injurious. The primary effects are headach, nausea, languor, loss of appetite and sleep; the secondary effects are manifested by a species of cachexia, indicated by an altered complexion. He attributes these symptoms to the nicotina which is volatilized. (Gaz. Méd. Mai. 3, 1845.)

Poisoning by tobacco has not often given rise to medico-legal discussion. This is the more remarkable, as it is an easily accessible poison to the lower classes, and the possession of it would not, as in the case of other substances, excite surprise or suspicion. It is probably more extensively used to aid the purposes of robbers than is commonly believed; and there is reason to suppose that porter and other liquors sold in brothels, are sometimes drugged with it. Dr. Ogston communicated an interesting case of this kind to Dr. Christison (Op. cit. 850,) in which tobacco was administered to a man in whiskey, and he soon afterwards died in a state of insensibility, without being able to give any account of the circumstances. Dr. Ogston detected *Nicotina* in the contents of the stomach. An investigation took place in this metropolis, in the autumn of 1847, in which a man was charged with attempting to poison his wife, by administering snuff in ale. The woman's life was saved by the speedy use of the stomach-pump. The case was dismissed, as there was a want of clear proof of criminal intention. A question here arose as to what quantity of tobacco would destroy life. The medical witness is reported to have said, that a quarter of an ounce infused in a pint of liquid, would be sufficient to destroy three persons. This is no doubt true. Thirty grains have proved fatal, and twenty grains might even kill an adult. Many kind of snuffs are, however, extensively adulterated with various powders; some contain lime, and even red lead: hence they are not to be regarded as consisting of pure tobacco.

Analysis.—Tobacco may be found in substance in an organic liquid, or in the stomach: it may then be recognised by its odour and physical properties. Its poisonous effects are due to the presence of a peculiar volatile alkaloid, which, like conia, is liquid. It is called Nicotina, and, according to Dumas, it forms less than the 1-1000th part of tobacco. Pereira assigns a much larger proportion than this:—thus, according to him, 1000 parts of Cuba tobacco give 8·64 of nicotina; of Virginia, 10·; and of smoking tobacco, 3·86. (Mat. Med. ii. 1247.)

Nicotina.—This liquid is commonly described as colourless; but it is more generally of a yellow colour, which becomes darker by exposure to air. It has a pungent, irritating odour, like that of tobacco, and an acrid burning taste.

It has an alkaline reaction, and is soluble in water. It remains liquid down to 21°. At 212° it evolves white alkaline vapours, which have a powerful odour. It is not very inflammable, but it may be burnt like an oil by means of a wick. It is a most powerful poison. A concrete volatile oil, called NICOTIANIN, is obtained by the distillation of tobacco leaves; and there is an empy-reumatic oil, which results from the decomposition of tobacco by distillation at a very high temperature.

Tobacco in organic liquids.—Nicotina may be thus extracted, so as to demonstrate its properties. Digest the evaporated residue in water, with diluted sulphuric acid, so as to dissolve the nicotina. Mix this with slaked lime, and distil carefully: nicotina mixed with ammonia passes over. Neutralize this liquid with diluted sulphuric acid,—concentrate and separate the sulphate of nicotina from that of ammonia by means of ether. The sulphuric acid may be separated by hydrate of baryta, and the nicotina obtained from the ether by distillation or spontaneous evaporation. (For other details, see Dumas, Traité de Chimie, t. v. 814.) By a process analogous to this, Dr. Ogston was enabled to discover it in the case of the individual whose body he inspected, (page 631.) A large quantity of tobacco must be present in order that the process should succeed, since *sixty grains* of common tobacco do not contain more than 0·24 gr., or less than a quarter of a grain, of nicotina. M. Schloesing has recently suggested another process for the extraction of this alkaloid. (Comptes Rendus, 21 Dec. 1846, 1142.)

YEW.

It has been long known that the berries and leaves of the yew-tree (TAXUS BACCATA) are poisonous to cattle;—they act very energetically, and produce death in a few hours, sometimes without vomiting or purging. It is stated by Dr. Percival, that a tablespoonful of the *fresh leaves* was administered to three children of five, four, and three years of age as a vermifuge. Yawning and listlessness soon succeeded; the eldest vomited a little, and complained of pain in the abdomen, but the other two suffered no pain. They all died within a few hours of each other. An interesting case of poisoning by the *berries* of this tree, was published a few years since by Mr. Hurt, of Mansfield. A child aged three years and a half, ate a quantity of yew-berries about eleven o'clock. In an hour afterwards, the child appeared ill, but did not complain of any pain. It vomited part of its dinner, mixed with some of the berries. A medical man was sent for, but the child died in convulsions before he arrived. On inspection, the stomach was found filled with mucus, and the half-digested pulp of the berries and seeds. There were patches of redness in the mucous membrane, and this was so much softened that it could be detached with the slightest friction. The small intestines were also inflamed.

In March, 1845, a case was reported to the Dublin Pathological Society by Dr. Mullan, in which a lunatic had died from the effects produced by yew-leaves. The deceased was observed chewing the plant, probably from that perversion of appetite so commonly observed in insanity, and before the attendants had taken it from him, he had succeeded in swallowing a portion of the masticated juice. He was soon afterwards seized with giddiness, sudden prostration of strength, vomiting, coldness of the surface, spasms, and irregular action of the heart. He died in fourteen hours. On inspection, the stomach was found much distended,—it contained some yew-leaves. There was emphysema in the submucous tissue, but no other abnormal change: there was some thickening with opacity of the arachnoid, which might have been due to the insanity. (Dub. Hosp. Gaz., May 15, 1845, p. 109.)

There is no doubt that the yew is a powerful poison of the narcotico-irritant class. The nature of the poisonous principle is unknown, nor is it certain whether, with respect to the berry, the poison is lodged in the pulp or the seed. Infusion of yew-leaves, which is popularly called yew-tree tea, is sometimes used for the purpose of procuring abortion by ignorant midwives. A case of death from a person drinking this infusion is reported in the registration returns for 1838-9. In the returns for 1840, there is also one death of a female, æt. 34, referred to her having eaten the berries of the yew.

Analysis.—The leaves or berries may be found in the stomach. The yew and the savin are the only poisons of the coniferous order which grow in this country. The apex of the leaf of the yew is lancet-shaped, that of the savin acuminated: the yew-leaf does not possess the powerful odour of savin when rubbed. Yew-berries are seen in autumn; they are of about the size of a pea, of a light red colour, dull on the surface, and translucent. They are open at the top, allowing a hard brown kernel to be seen. This is of an ovoid shape, and it forms the greater part of the berry. The fine red skin contains a colourless and remarkably viscid or adhesive juice, which reddens litmus paper, and has a nauseous sweetish taste.

POISONS NOT ARRANGED IN THE FOREGOING CLASSES.

Indian Hemp, Cannabis Sativa vel Indica.—The inspissated resinous exudation of the leaves and stems of this plant, known in the East as Churrus and Chaschisch, has been introduced into this country as a substitute for opium. Its effects appear to be very uncertain: in some instances, large doses, either in the form of extract or tincture, have been given with impunity, while in other cases, symptoms resembling those of narcotic poisoning, have resulted from small quantities. On the whole, its effects in England have been much less powerful that those observed in India. In a large dose, Indian hemp produces a pleasant species of intoxication. The nervous system is also most singularly affected, while the intellectual powers remain unaltered. According to Mr. Ley, the inebriation is of the most cheerful kind, causing the person to sing and dance, and to eat food with great relish. It also excites aphrodisiac propensities. The intoxication, which lasts about three hours, and is sometimes attended with uncontrollable laughter, is succeeded by sleep. There is no nausea, sickness, or diarrhœa; and the day following, there may be slight giddiness, with vascularity of the eyes. (Prov. Med. Jour., March 18, 1843, p. 487.) Dr. O'Shaughnessy found that a grain produced insensibility after the stage of excitement, and that the individual was perfectly cataleptic. Mr. Donovan was induced to try the effects of it upon himself, and he swallowed fourteen grains of the resinous extract at night. He awoke about five o'clock in the morning, and felt a rush of strange sensations through his head, attended with a cracking noise and a sense of vibration through the whole of his body. An explosion then appeared to take place in his head, and he gradually lost all sense of feeling. His intellect was not in the least disturbed,—his memory was good,—he could reason clearly, and was as conscious of external objects as in health; but he had absolutely lost the consciousness of having a body: his whole corporeal existence appeared to be comprised within his head and in a small portion of his chest near his throat! In these parts he felt as much alive as ever; but all other parts were without feeling, and apparently annihilated. These extraordinary sensations gradually passed off: he became sick, and on attempting to get out of bed, he could scarcely walk for giddiness. (Dub. Med. Press, March 5, 1845, 153.) The following case will show that the administration of this medicine is not unattended with danger. A lady, æt. 26, by the advice of her medical attendant, swallowed three doses, of five drops in each, of tincture of Cannabis Indica, with an interval of two hours between the first and second dose, and three hours between the second and third. She joined her family at dinner as usual; but some incoherence of manner and speech was observed, and almost immediately afterwards, she became violently sick, and vomited. She was quite unconscious; the extremities and body were cold; the wrist pulseless; eyes wide open and staring,—pupils somewhat contracted, and quite insensible to the strongest light, with violent convulsions of the whole frame, and involuntary twitchings of the muscles. These latter symptoms lasted for two days, whether she was asleep or awake. She was quite insensible for a quarter of an hour, and did not recover her consciousness during the whole night. The pulse, which varied from 140 to 150, was extremely feeble, and intermitted from time to time during the two following days. The patient had not been previously subject to fits of any description. (Monthly Jour. Med. Sciences, April 1847, p. 776; and Prov. Med. Jour. March 10, 1847.) If this drug should come into more extensive use, it is not unlikely that it may give rise to serious accidents. It appears to be very uncertain in its effects.

Quinia.—This alkaloid, obtained from Peruvian bark, is extensively used in medicine under the form of disulphate, or as it is commonly called sulphate of quinine. Some alarming effects have of late years been observed to follow its administration in large, or in small doses frequently repeated. In doses of from ten to twenty grains, Dr. Pereira states that it causes griping pain and heat in the abdomen, with vomiting and purging, ptyalism, a febrile condition of the system, headach, giddiness, somnolency, delirium, and stupor. (Mat. Med. ii. 1407.) In addition to these symptoms, amaurosis and convulsions have been observed in certain cases. The common medicinal dose is from three to ten grains; but even this cannot always be borne by patients. M. Desiderio has observed that this substance produced, both in man and animals, drowsiness, difficulty of maintaining the erect position, obscured vision, and drooping of the eyelids. He regards it as a poison, and considers that, in its toxicological effects, it resembles the acetate of morphia. M. Sandri found that eighteen grains killed a rabbit in less than three hours. On inspection, the whole of the brain and its membranes were strongly congested; the lungs were of a bright red colour, and also congested; and the heart contained a large quantity of blood brighter than natural. (Gaz. Med. 17 Juillet, 1847, 585.) Instances of its fatal operation in the human subject are not wanting, as the following cases will show. A man, æt. 26, labouring under acute rheumatism, was ordered by M. Recamier, at the Hôtel Dieu, to take on the first day, forty-six grains of sulphate of quinia in twelve powders, one to be taken every hour. On the second day seventy-seven grains were prescribed in twelve powders, to be taken hourly. The patient had taken nearly two-thirds of the quantity, when he was suddenly attacked with delirium, and died in a few hours. On dissection, the membranes of the brain were found inflamed, and there was sanguineous effusion. (Med. Gaz. xxxii. 430.) Dr. Baldwin, of the United States, has reported a case in which convulsions, blindness, and death followed from the administration of this medicine to a girl five years of age. Two grains were given every second hour. On the following day there was great anxiety of countenance, with irregular heavy breathing, and extreme restlessless. After a certain period, a dose of four grains was given twice at an interval of three hours. The restlessness increased, the pupils of the eyes were widely dilated, and she was found to be totally blind. She had violent convulsions, and died three hours after taking the last dose. The principal appearance was great vascularity of the stomach and intestines. Dr. Baldwin considers from his observations, that with this will be found congestion of the brain and lungs, and a dark and fluid condition of the blood. He believes, that from *fifty to eighty grains* of a pure specimen of quinine given at once, would produce death nine times out of ten in healthy adults; and occasionally even smaller doses might operate fatally. (Amer. Med. Jour. Med. Sci., April 1847, p. 292.) In the same journal (p. 515,) four cases are related by Dr. M'Lean, in which blindness was the result of large doses of this medicine. In a set of cases reported by Dr. Thom, the symptoms partook of the characters of mercurial erethism. (Philadelphia Med. Examiner, April 1847, 217.)

There can be very little doubt that quinia, like the other alkaloids, is absorbed. It is stated by Landerer that it was found in the milk of a nurse, to whom the sulphate had been administered medicinally, and that the child refused to take the milk. On the whole the subject of poisoning by quinine has received but little attention in this country. A sufficient number of facts has, however, been accumulated in France and America, to show that this medicine cannot be administered in large doses, or for a long continuance, with impunity.

Treatment.—This consists in the free administration of any liquid containing tannin, as this substance forms a completely insoluble compound with the alkaloid, and may thus retard absorption. This treatment should be followed by emetics, or the use of the stomach-pump.

Analysis.—The common sulphate of quinine crystallizes in light flocculent prisms. It is not very soluble in cold water, except on the addition of an acid, when it forms a solution which, even when very largely diluted with water, presents an intensely blue colour at the surface (a characteristic property.) Its solution is precipitated by caustic potash, and the precipitate is not soluble in an excess of the alkali;—also abundantly by infusion of galls. Sulphuric acid is detected in the liquid by the process elsewhere described. (See SULPHURIC ACID.) The crystals, when pure, are not carbonized by strong sulphuric acid in the cold: when exposed to heat, on platina foil, they melt and burn like a resin.

CARBAZOTIC ACID (NITROPICRIC ACID.)—This is a solid crystalline acid, obtained from the action of nitric acid on indigo. According to the results of experiments on animals, it appears to be a narcotico-irritant poison. Ten grains have sufficed to kill a dog in less than two hours. The symptoms consisted in tremor of the limbs, stupor, and convulsions. After death the stomach was found dyed by the acid of an intense yellow colour; and as a proof that it had been absorbed, the same colour was found to extend through the coats of the blood-vessels.

Independently of the vegetable poisons hitherto considered, a narcotico-irritant action is possessed by the ANAGALLIS ARVENSIS (Meadow pimpernel,) MERCURIALIS PERENNIS (Mountain mercury,) CHÆROPHYLLUM SYLVESTRE (Wild chervil,) SIUM LATIFOLIUM (Procumbent water-parsnip,) KALMIA LATIFOLIA (Mountain laurel,) SPIGELIA MARYLANDICA (Pink root,) ARISTOLOCHIA CLEMATIS (Common birthwort,) and the SAPONARIA OFFICINALIS, or Soapwort. The poisonous principle in the last-mentioned plant is SAPONINE; and, according to M. Malapert, it exists in numerous other vegetables, and confers upon them noxious properties. (Gaz. Méd., Jan. 30, 1847, 98.)

POISONOUS GASES.

CHAPTER XLIII.

MODE OF ACTION OF THE POISONOUS GASES—AMMONIA—CARBONIC ACID—SYMPTOMS AND APPEARANCES—ITS OPERATION AS A POISON—TREATMENT—ANALYSIS—CHARCOAL-VAPOUR—FATAL EFFECTS OF COAL-VAPOUR—SULPHUROUS ACID—VAPOUR OF LIME- AND BRICK-KILNS—CONFINED AIR—COMBUSTION IN MIXTURES OF CARBONIC ACID—DIFFUSION OF THE GAS IN AIR—COAL-GAS—CARBURETTED HYDROGEN—CARBONIC OXIDE—EXHALATIONS OF THE DEAD—CHLORINE—MURIATIC ACID—PROTOXIDE AND DEUTOXIDE OF NITROGEN—OXYGEN—CYANOGEN AND OTHER GASES.

General remarks.—The numerous *gases* with which chemists are acquainted, are found to vary materially in their operation when introduced into the lungs; and a division has been established among them into those which have a *negative*, and into those which have a *positive* action. The former alone can be considered to cause death by asphyxia or *suffocation;* for the gases which have a positive influence, must be regarded as poisons. Now experiment has shown that there are but two gases which are essentially negative in their operation,—these are HYDROGEN and NITROGEN; all the others have a poisonous action when introduced into the body. Indeed, with regard to HYDROGEN, some doubt may be fairly entertained respecting its claim to be considered as a truly negative agent; for the researches of Allen and Pepys in this country, and the observations of Wetterstedt in Sweden, have shown that it cannot be substituted for nitrogen in atmospheric air, without inducing somnolency and lethargy. (Berzelius, Traité de Chimie, vii. 106.) If, then, we admit that the greater number of the gases are poisonous, it is scarcely correct to regard these bodies as purely asphyxiating agents. The state of lifelessness which follows their introduction into the lungs, is not to be ascribed to the simple negation of air, as in the case of drowning, hanging, or strangulation; but to a deleterious impression produced on the system, something analogous in its effects to that which is observed to follow the ingestion of a poisonous dose of hydrocyanic acid. The differences are, that the poison is aërial, acts more rapidly, and is applied to the surface of the lungs instead of the stomach; but, strictly speaking, a person is no more suffocated by carbonic acid, than he is by arsenuretted hydrogen. (See ante, p. 308.)

The greater number of the poisonous gases are never likely to be met with in the atmosphere so abundantly as to produce injurious consequences; they are chiefly complex products of art:—hence fatal accidents, arising from their inhalation, most commonly occur under circumstances which can leave no question respecting the real cause of death. Agents of this description can rarely be employed with any certainty as instruments of murder; and if they were so employed, the fact could be established only by circumstantial evidence. One alleged instance of murder by carbonic acid is reported by M. Devergie.

(Ann. d'Hyg. 1837, i. 201.) Death, when arising from the respiration of any of the gases, is generally attributable to suicide or accident. In France, it is by no means uncommon for individuals to commit self-destruction by sleeping in a closed apartment, in which charcoal has been suffered to burn; while in England, accidental deaths are sometimes heard of, where coal has been employed as fuel in small and ill-ventilated rooms. On such occasions, a person may be found dead without any apparent cause to the casual observer,—the face may appear tumid and discoloured, and the cutaneous surface may be covered with ecchymosed patches. The discovery of a body under these circumstances, will commonly be sufficient, in the eyes of the vulgar, to create a suspicion of murder; and some individual, with whom the deceased may have been at that period on bad terms, will, perhaps, be pointed out as the murderer. In such a case, it is obvious that the establishment of the innocence of an accused party may depend entirely on the discrimination and judgment of a medical practitioner. An instance, illustrative of the consequences of this popular prejudice, occurred in London in 1823. Six persons were lodging in the same apartment where they were all in the habit of sleeping. One morning an alarm was given by one of them, a female, who stated that on rising she found her companions dead. Four were discovered to be really dead, but the fifth, a married man, whose wife was one of the victims, was recovering. He was known to have been on intimate terms with the female who gave the alarm, and it was immediately supposed that they had conspired together to poison the whole party, in order to get rid of the wife. The woman who was accused of the crime was imprisoned; and an account of the supposed murder was soon printed and circulated in the metropolis. Many articles of food about the house were analyzed, in order to discover whether they contained poison, when the whole of the circumstances were explained by the man stating that he had placed a pan of burning coals between the two beds before going to sleep and that the doors and windows of the apartment were closed. (Christison, 583.) A set of cases of a similar kind, in which there was at first a very strong suspicion of poisoning, has been lately reported in the Medical Gazette, by Mr. Smith, of Liverpool. (xxxvi. 937.)

AMMONIA.

The effects of this irritant gas have already been described in speaking of its operation as a poison when dissolved in water, (ante, p. 224.) Animals when immersed in it are speedily destroyed under symptoms similar to those caused by the solution. The whole of the body is strongly impregnated with the powerful and well-known odour of ammonia.

CARBONIC ACID.

This gas is freely liberated in respiration, combustion, and fermentation; it is also extricated in the calcination of chalk or limestone, and is abundantly diffused through the shafts and galleries of coal-mines, where it is commonly called choke-damp. Carbonic acid gas is likewise met with in wells, cellars, and other excavations in the earth. In these cases it is found most abundantly generally on the soil, or at the lower part of the well; and it appears to proceed from the decomposition of animal aad vegetable matters confined in such situations. The slow evaporation of water strongly charged with the gas, while trickling over the sides of these excavations, may likewise assist in contaminating the air. Damp sawdust or straw slowly absorbs oxygen from a confined atmosphere, and sets free carbonic acid.

SYMPTOMS.—The symptoms of poisoning by this gas, will vary according to the degree of concentration in which it is present in the atmosphere respired. When it exists in a fatal proportion, the symptoms commonly observed are as follows:—a sensation of great weight in the head, giddiness, a sense of constriction in the temporal regions, a ringing in the ears, with a pungent sensation in the nose; a strong tendency to sleep, accompanied by vertigo, and so great a loss of muscular power, that if the individual be at the time in an erect posture, he instantly falls as if struck to the ground. The respiration, which is observed to be at first difficult and stertorous, becomes suspended. The action of the heart, which on the first accession of the symptoms is very violent, soon ceases. Sensibility is lost, and the person now falls into a state of profound coma, or apparent death. The warmth of the body still continues; the limbs remain flexible, but they have been observed to become rigid, or even occasionally convulsed. The countenance is commonly of a livid or of deep leaden colour, especially the eyelids and lips, but on some occasions it is stated to have been pale. The access of these symptoms has been sometimes accompanied by a pleasing sensation of delirium, while at others the most acute pains have been suffered. In some instances there appears to have been irritability of the stomach; for the affected person has ejected the contents of the stomach in a semi-digested state. Those who have been resuscitated, have often felt pain in the head, or pain and soreness over the body for several days; while, in a few severe cases, paralysis of the muscles of the face has supervened on recovery.

Post-mortem appearances.—Externally, the whole of the body appears as if it were swollen, especially the face, which is generally livid, and the features are much distorted. The cutaneous surface is covered in parts by patches of a violet hue, but, in some instances, the skin has been extremely pale; the eyes are generally prominent, and, in many cases, retain their usual brilliancy for some time after death. The body of an individual who has perished from the inhalation of carbonic acid, is said to retain the animal heat, cæteris paribus, for a longer period than usual; and, hence, according to Orfila, cadaverous rigidity does not commonly manifest itself until after the lapse of many hours. In a case to be related presently, the body was, however, found to have cooled considerably within the short space of two hours. On making a post-mortem inspection, the venous system is found filled with blood of a dark colour; and the vessels of the lungs and brain are observed to be especially in a state of congestion. The tongue appears swollen, and it is stated by Orfila, that the mucous membrane of the intestinal canal is often interspersed with dark ecchymosed patches.

It will be seen that there is nothing very characteristic in the post-mortem appearances, and thus it is always easy to ascribe death to apoplexy or some other cause; but it should be remembered that carbonic acid itself acts by inducing apoplexy or cerebral congestion. A stove was actually allowed to be patented a few years since—the principle of which was to permit the escape of the products of combustion in an invisible form into an apartment! There were many educated persons so ignorant as to believe, that because the fumes were invisible they were inert; others speculated upon the quantity of carbonic acid evolved being *small!* The use of this stove appears to have led to the death of a man named Trickey, in St. Michael's church, in 1838, and many other serious accidents. The case of *Trickey* is in many respects worthy of the attention of the medical jurist. (See Lancet, Nov. 1838.)

Action on the body.—Some difference of opinion still exists respecting the manner in which carbonic acid acts on the body. Sir Humphry Davy ascertained that carbonic acid, in a perfectly pure state, did not pass into the trachea when an attempt was made to respire it: the glottis seemed to close spasmodically at the moment that the gas came in contact with it. On diluting the carbonic acid with about twice its volume of air, he found that he could breathe it; but it soon produced symptoms of vertigo and somnolency. In fact, in a diluted state, it is certain that it must penetrate into the lungs, or otherwise it would be impossible to explain why it should produce any other symptoms than those witnessed in the inhalation of hydrogen or nitrogen. The facts which have been collected by Dr. Christison show, in a striking point of view, that carbonic acid is a real and energetic poison of the narcotic kind. If, as Nysten supposed, it had a negative effect when respired, it ought to follow, that it might be substituted for nitrogen, in the proportion in which that gas exists in atmospheric air. But a mixture of carbonic acid and oxygen, in atmospheric proportions, has been shown by M. Collard de Martigny, to produce rapidly fatal effects upon the animal system. Such a mixture cannot be breathed even for a period of two minutes, without giving rise to serious symptoms.

When the gas enters into the pulmonary cells, it is probably *absorbed* by the blood, and circulated with that fluid throughout the body. Its specific action on the brain may be inferred from the headach, vertigo, somnolency, and coma, which follow its introduction, as also from the loss of muscular power in persons labouring under its effects, and the paralysis which is sometimes seen in those who have recovered. A very small proportion of carbonic acid, when respired for a certain time in combination with air, will suffice to destroy life in man, or in any of the higher orders of animals. It is generally admitted by physiologists, that an atmosphere containing more than *one-tenth* of its volume of carbonic acid, will, if introduced into the lungs, speedily prove fatal to human life. M. Guérard has lately called in question the general opinion that carbonic acid is very fatal to life. He says it may be mixed in very large proportions with atmospheric air without causing death, and attributes the noxious effects of charcoal-vapour to carbonic oxide, which he says will prove fatal when in the proportion of only four or five per cent. (Ann. d'Hyg. 1843, ii. 54.) If M. Guérard had extended his experiments to the Grotto del Cane, at Pozzuoli, near Naples, he would have found that mixtures which he describes as innocent, are speedily fatal to animal life. The air of the grotto is a mixture of carbonic acid, common air, and aqueous vapour: it contains no carbonic oxide; and I have not only witnessed its fatal effects on animals, but have myself experienced the incipient symptoms of poisoning by carbonic acid from respiring it.

It is necessary in these cases to make a distinction between the contamination of air from the admixture of free carbonic acid, and the case where the carbonic acid is formed by combustion or respiration in a close apartment, at the expense of the oxygen actually contained in air. Every volume of carbonic acid formed by combustion, indicates an equal volume of oxygen removed. Such an atmosphere is, *cæteris paribus*, more destructive than another where the air and gas are in simple admixture. If we assume that in each case the noxious atmosphere contains ten per cent. of carbonic acid, then in one instance there will be seven per cent. more of oxygen, and seven per cent. less of nitrogen, than in the other, since the production of ten parts of carbonic acid implies the loss of ten parts of oxygen.

This difference in the proportions may not be, practically speaking, correct; because there is no apartment sufficiently closed, to prevent air rushing in from the exterior while combustion is going on within it; but, nevertheless, the above statement may be taken as an approximation to the truth. When the gas is respired in the lowest poisonous proportion, the symptoms come on more slowly, and the transition from life to death is frequently tranquil; this is what we learn from the histories of suicides. The symptoms in such cases appear to resemble closely those which indicate the progressive influence of opium, or any other narcotic poison, on the body.

TREATMENT.—The best means for resuscitation are the employment of cold affusion, with stimulating embrocations to the chest and extremities. If the surface be cold, a warm bath should be employed, and on the appearance of any signs of recovery, venesection may be performed. If at hand, oxygen gas may be introduced into the lungs. A case, in which the use of this gas is said to have been successful, is quoted in the Lancet, July 26, 1844, 531. Oxygen gas was used for this purpose nearly forty years ago, by the late Dr. Babington. (Med. Chir. Trans. i. Art. 8.)

ANALYSIS.—Sometimes a medical jurist may be required to state, for the purposes of justice, the nature of the gaseous mixture in which a person may have died. He will have but little difficulty in determining whether carbonic acid is the deleterious agent in such a mixture. When it exists in a confined atmosphere, its presence may be identified, if previously collected in a proper vessel, by the following characters. 1. It extinguishes a taper if the proportion be above twelve or fifteen per cent.; and from the extreme density of the gas, the smoke of the extinguished taper may be commonly seen to float on its surface. 2. Lime-water, or a solution of subacetate of lead, is instantly precipitated white when poured into a jar of the gas; and the precipitate thus formed may be collected by filtration, and proved to possess the well-known properties of carbonate of lime or lead. Air containing only one per cent. of carbonic acid, scarcely affects lime water. 3. When a solution of chloride of lime, coloured by litmus, is added, the blue colour, on agitating the liquid in the gas, is discharged. This clearly distinguishes carbonic acid from nitrogen. The *proportion* in which carbonic acid exists in a mixture, may be determined by introducing into a given quantity, in a graduated tube over mercury, a strong solution of caustic potash. Absorption will take place after a certain time, and the degree of absorption will indicate the proportion of carbonic acid present. When this destructive agent exists in a confined spot, as in a well or cellar, it may be generally got rid of by placing within the stratum a pan containing the hydrate of lime, loosely mixed into a paste with water, by exciting combustion at the mouth of the pit, or, what is better, where available, by a jet of high-pressure steam. Lives are often successively lost on these occasions in consequence of one individual descending after another, in the foolish expectation of at least being able to attach a rope to the body of his companion. The moment that the mouth falls within the level of the stratum, all power is lost, and the person commonly sinks lifeless. The gas may be collected by lowering a bottle filled with fine sand by means of a string attached to the neck, and guiding the bottle by another string attached to its base. When the bottle is within the stratum, it should be turned with its mouth downwards, then rapidly raised with its mouth upwards, by pulling the string attached to the neck.

CHARCOAL-VAPOUR.

The gas extricated during the combustion of Charcoal, according to the experiments of Orfila, is not pure carbonic acid, but a very compound mixture. It operates fatally when respired, chiefly in consequence of carbonic acid contained in it, the proportion of which, however, is subject to variation, according to whether the combustion be vivid or not. When the charcoal burns vividly, the quantity of carbonic acid is said to be less than when it is either nearly extinguished or beginning to burn. In the former case, the carbonic acid is in the proportion of about eleven per cent. by volume—in the latter, the proportion amounts to about fourteen per cent.; the remainder of the mixture is made up of air, of free nitrogen, and of a portion of carburetted hydrogen, if the charcoal be not too intensely ignited. (Orfila.)

The following case, illustrating the effects of charcoal-vapour, has been reported by Mr. Collambell. (Med. Gaz. xxvii. 693.) In January, 1841, a man was engaged to clean the windows of three small rooms on the basement-story of a house. The first room had a door opening into a court-yard—the others merely communicated with each other by a central door, and there was no fire-place in any one. A brazier of burning charcoal had been placed in the outer room for the purpose of drying it, but it appeared that the deceased had shut the outer door and had removed the brazier into the inner room of the three, leaving the communicating doors open. In two hours the man was found quite dead, lying on the floor of the middle room. The countenance was pale, as well as the whole of the skin; the eyes were bright and staring, the pupils widely dilated; the lips exsanguine; the jaw firmly fixed; the tongue protruding, and the face and extremities cold. Some frothy mucus had escaped from the mouth. The person who discovered the deceased, found the ashes in the brazier still burning, and he experienced great oppression in breathing. An inquest was held without an inspection, and a verdict of accidental death returned. The body was afterwards privately inspected by Mr. Collambell. On opening the head, the vessels on the surface of the brain were found highly distended with dark liquid blood; the pia mater was bedewed with serum. The brain was of unusually firm consistence, and numerous bloody points appeared on making a section of it. The lateral ventricles were distended with about an ounce and a half of pale serum, and the vessels of the plexus choroides were much congested. The cerebellum was firm, and presented on section numerous bloody points. About two ounces of serum, tinged with blood, were collected from the base of the skull. The lungs had a slate colour. On the left side of the chest there were eight ounces of serum, tinged with blood, and nearly an equal quantity on the right side. On cutting into the organs, a large quantity of serous fluid, mixed with blood, escaped. The bronchial tubes were filled with a frothy fluid, tinged with blood. The pericardium contained an ounce of pale serum: the heart was enlarged; the cavities contained no blood: the liver and kidneys were, however, much gorged. There was no doubt that the cause of death was the inhalation of carbonic acid; and it is probable that the man died from respiring but a comparatively small proportion. The capacity of the chambers must have nearly reached two thousand cubic feet; the deceased had been there only two hours, and when the person who discovered him entered the rooms, the air was not so vitiated but that he could breathe, although with some oppression. The fuel was then in a state of combustion.

It often excites surprise on these occasions that no exertion is made to escape, when it would apparently require but very slight efforts on the part of the individual. The fact is, that the action of the vapour is sometimes very insidious; one of its first effects is to create an utter prostration of strength, so that even on a person awake and active, as in the case just related, the gas may speedily produce a perfect inability to move or to call for assistance. For some good remarks on the action of charcoal vapour by Dr. Bird, see Guy's Hospital Reports, April 1839; and for a case illustrative of the dangerous effects of the diluted vapour, see Ed. Med. and Surg. Jour. i. 541. In this instance, a charcoal brazier was left only for a short time in the cell of a prison. It was removed, and the prisoners went to sleep. They experienced no particular effects at first, but after some hours, two were found dead. Thus, then, an atmosphere which can be breathed for a short time with impunity, may ultimately destroy life.

In a case of alleged murder by carbonic acid, which occurred in Paris a few years since, a question was put to the medical witnesses, respecting the *quantity of charcoal* required to be burnt in a particular chamber in order to asphyxiate two adult individuals. (Ann. d'Hyg. 1837, i. 201; 1840, 176: also Brit. and For. Rev. xi. 240, and xxiii. 264.) This question could of course only be answered approximately; because in burning charcoal, the sole product is not carbonic acid, and the substance itself is by no means pure carbon. Then again, much of the carbonic acid formed, may escape in various ways from an imperfectly closed apartment. An attempt was made to infer the quantity of charcoal consumed, from the weight of ashes found in the apartment; but no satisfactory answer could be given to this question. The prisoner was, however, convicted of murdering his wife by carbonic acid.

M. Devergie has shown that the slow combustion of *wood* may lead to the evolution of a noxious vapour and give rise to dangerous consequences (Ann. d'Hyg. 1835, i. 442.) His remarks have been recently confirmed by two cases published by MM. Bayard and Tardieu. A man and his wife were found dead in bed. There was a smoky vapour in the apartment, but no fire had been lighted in the grate, and the chimney was blocked up. The planks of the floor were widely separated, and there was a large hole in the boards at the foot of the bed communicating with the apartment below. It was found, on examination, that some joists connected with the flue of an iron plate, which had been heated for making confectionary the previous day, were in a smouldering state; that the vapour had entered the bedroom of the deceased through the crevices in the floor, and not finding a vent by the chimney, had led to these fatal results. It is remarkable that the source of combustion was nearly nine yards distant, and one person, who slept nearer to the flue of the iron plate, entirely escaped. In the husband, the skin was of a reddish tint, the blood liquid, the cavities of the heart empty, the lungs gorged, and there was no subpleural ecchymoses. In the wife, there was less redness of the skin, the blood was coagulated in the cavities of the heart principally on the right side extending to the vessels; less engorgement of the lungs, and a great number of subpleural ecchymoses, indicating that strong efforts had been made to respire. There was at first a rumour of poisoning, which was only removed by a close examination of the locality. (Ann. d'Hyg. Oct. 1845, 369.)

COAL VAPOUR. SULPHUROUS ACID.

The gases extricated in the smothered combustion of coal are of a compound nature. In addition to carbonic acid, we may expect to find in the atmosphere of a close room, in which such a combustion has been going on, SULPHUROUS ACID GAS, and the sulphuretted and carburetted hydrogen gases. These emanations are equally fatal to life; but in consequence of their very irritating properties, they give warning of their presence, and are therefore less liable to occasion fatal accidents. The sulphurous acid gas, when existing in a very small proportion in air, has the power of irritating the glottis so violently, that, if accidentally respired, it would commonly compel the individual to leave the spot, before the vapours had become sufficiently concentrated to destroy life. Nevertheless, accidents from the combustion of coal sometimes occur.

The following cases will convey a knowledge of the symptoms and post-mortem appearances which are commonly met with on these occasions. Some years since, four individuals, in a state of asphyxia, were brought to Guy's Hospital. It appeared that on the evening before, they had shut themselves up in the forecastle of a coal-brig, and had made a fire. About six or seven o'clock the same evening, some of the crew accidentally placed a covering over the flue on the outside, and thus stopped the escape of smoke from the fire, which was made of a kind of coal containing much sulphur. Early in the morning, one of the crew, on opening the hatches, observed three of the inmates lying on the floor senseless, and frothing at the mouth; the fourth in his crib, in a similar condition. The air in the place was most offensive. After the men had been brought on deck, one of them, aged twenty-one, began to recover, and when brought to the hospital, he seemed only giddy, as if intoxicated. He soon completely recovered. Another, aged forty, after breathing oxygen gas, and having brandy and ammonia exhibited, showed no symptom of recovery, and died in a few hours. A third, aged seventeen, soon began to rally, and, in a few hours, was perfectly enabled to answer questions; he declared that he felt no pain, sense of oppression, or weight either in his head or chest. The fourth, aged fifteen, died the following day, having exhibited no symptoms of rallying. Stimulants were administered internally, and warm fomentations were used, but all efforts to produce reaction failed. The appearance of the individuals when brought in, were as follows:—lips purple, countenance livid, surface of the body cold, hands and nails purple, respiration very quick and short; pulse small, quick, and feeble; pupils fixed; and total insensibility. The body of the man aged forty was inspected about four hours after death. The membranes of the brain were congested, and there was a large quantity of fluid under the tunica arachnoides. The sinuses were gorged with blood. The lungs were in a state of great congestion, as also the right cavities of the heart. It was remarked, that this corpse was similar in appearance to that of an executed culprit. The body of the lad aged fifteen was inspected about thirty-three hours after death. Under the pia mater was observed one small ecchymosed spot; in the substance of the brain there were more bloody points than usual; a small quantity of fluid was found under the tunica arachnoides, and the sinuses were full of coagulated blood. The lungs showed no congestion, but the right cavities of the heart were much distended with blood. (For an account of two cases of recovery from the effects of coal-vapour, see Med. Gaz., ix. 935.)

An interesting case of the fatal effects of coal-vapour has been lately published by Dr. Davidson. The man lost his life from sleeping in a closed room with a fire to which there was no flue. The lungs were found gorged with blood, the trachea and bronchi filled with a frothy muco-sanguineous fluid; and the mucous membrane beneath was slightly injected. There was a small effusion in each pleural cavity. The right side of the heart was full of dark liquid blood. The dura mater was much injected; the sinuses of the brain, and the veins of the pia mater, were completely congested, and there was subarachnoid effusion. The substance of the brain when cut, presented numerous bloody points. (Month. Jour. April 1847, 763.)

[Many cases of asphyxia occur in this country from the effects of the gas of Anthracite coal; this is a compound resembling that from bituminous coal, but with less of the sulphurous acid gas.—G.]

ANALYSIS.—Sulphurous acid is immediately known by its powerful and suffocating odour, which resembles that of burning sulphur. The best test for its presence is a mixture of iodic acid and starch, which speedily acquires a blue colour when exposed to the vapour.

VAPOUR OF LIME- AND BRICK-KILNS.

In the burning of *lime*, carbonic acid is given out very abundantly, and in a pure form. It has been owing to the respiration of the gas thus extricated, that persons who have incautiously slept in the neighbourhood of a burning lime-kiln during a severe winter, have been destroyed. The discovery of a dead body in such a situation, would commonly suffice to demonstrate the real cause of death; but a practitioner ought not to be the less prepared to show that there existed no other apparent cause of death about the person. It is obvious that an individual might be murdered, and his body placed subsequently near the kiln by the murderer, in order to avert suspicion. If there be no external marks of violence, the stomach should be carefully examined for poison; in the absence of all external and internal lesions, medical evidence will avail but little; for a person might be criminally suffocated, and his body, if found under the circumstances above stated, would present no appearances upon which a medical opinion could be securely based. An accident is related by Foderé to have occurred at Marseilles in 1806, where seven persons of a family were destroyed in consequence of their having slept on the ground-floor of a house, in the court-yard of which a quantity of limestone was being burnt into lime. They had evidently become alarmed, and had attempted to escape; for their bodies were found lying in various positions. The court-yard was enclosed, and the carbonic acid had poured into the apartment through the imperfectly-closed window and door. In November 1838, a man died three days after being exposed to the vapours of a lime-kiln. (G. H. Rep. April 1839.) The vapour of a brick-kiln is equally deleterious, the principal agent being carbonic acid, although I have found that ammonia and muriatic acid are also abundantly evolved. In September 1842, two boys were found dead on a brick-kiln near London, whither they had gone for the purpose of roasting potatoes. Although the cause of death in the two cases was clearly suffocation, in one instance the body was extremely livid, while in the other there was no lividity whatever! Such accidents are very frequent. In November 1844, an inquest was held at Manchester on the body of a man who had died under similar circumstances.

CONFINED AIR.

An animal confined within a certain quantity of air, which it is compelled to respire, will soon fall into a state of lifelessness. A human being in the same way may be suffocated, if confined in a close apartment where the air is not subject to change or renewal, and this effect is hastened when a number of persons are crowded together in a small space. The change which air, thus contaminated by respiration, undergoes, may be very simply stated. The quantity of nitrogen in a hundred parts will remain nearly the same, the quantity of oxygen will probably vary from eight to twelve per cent., while the remainder will be made up chiefly of carbonic acid. Such air will also have a high temperature, if many persons are crowded together, and will be saturated with aqueous vapour containing animal matter poured out by the pulmonary and cutaneous exhalents. From this statement, it is evident that air which has been contaminated by continued respiration, will operate fatally on the human system, partly in consequence of its being deficient in oxygen, and partly from the deleterious effects of the carbonic acid contained in it. The proportion in which carbonic acid exists in respired air, must be subject to great variation; according to the experiments of Allen and Pepys, it never exceeds ten per cent. by volume of the mixture, how frequently soever it may have been received into and expelled from the lungs. Dalton found that the air in crowded rooms contained about one per cent. of carbonic acid, the atmospheric proportion being therefore increased tenfold. It is certain that insensibility and death would ensue in a human adult, before the whole of the oxygen of the confined air had disappeared; but the opportunity can rarely present itself of analyzing such a contaminated mixture, and hence it is impossible to specify the exact proportion in which carbonic acid would exist, when the confined air had proved fatal to persons who had respired it. M. Lassaigne has shown by direct experiment, that the carbonic acid in the air of close rooms is not collected on the floor, but equally diffused throughout. The whole mass of air is in fact vitiated, and requires renewal. (Med. Gaz. xxxviii. 351.)

Combustion in mixtures containing carbonic acid.—In reference to poisoning by carbonic acid, there is one circumstance which requires attention. It is a matter of very popular belief, and, in fact, it is generally asserted by writers on asphyxia, that the burning of a candle in a suspected mixture of carbonic acid and air, is a satisfactory proof that it may be respired with safety. Recent observations have, however, tended to show that this statement is not to be relied on as affording an indication of security. A case is related by Dr. Christison, where a servant, on entering a cellar in which grape-juice was fermenting, was suddenly seized with giddiness. She dropped her candle on the floor, but had time to leave the cellar and shut the door behind her, when she fell down senseless. Those who went to her assistance found, on opening the door, that the candle was still burning. Another case is referred to, where, in an attempt at suicide, on entering the apartment, the person was discovered to be in a state of deep coma, while the pan of charcoal was still burning; and in an instance just now reported, the same fact was observed (p. 642.) The results of some experiments on this subject have led me to the conclusion that a candle will burn in air which is combined with even ten or twelve and a half per cent. of its volume of carbonic acid gas: and although such mixtures might not prove immediately fatal to man, yet they would soon give rise to giddiness, vertigo, insensibility, and ultimately death, in those who, after having been once immersed in them, did not hasten to quit the spot. In air containing a smaller proportion than this,—five or six per cent.,—a candle will readily burn; but it is probable that such a mixture could not be long respired without causing fatal symptoms: hence the *burning of a candle can be no criterion of safety* against the effects of carbonic acid. It is perfectly true that, in gaseous mixtures where a candle is extinguished, it would not be safe to venture; but the converse of this proposition is not true, namely, that a mixture in which a candle burns, may be always respired with safety.

Diffusion of carbonic acid.—Of late years some important medico-legal questions have arisen, relative to the diffusion of this gas in air, when produced by combustion. It has been supposed that, owing to the great specific gravity (1·527,) it would collect on the floor of an apartment, would gradually rise upwards, and suffocate individuals at different times, according to the level on which they might be placed. Questions on this point have been variously answered, and great difference of opinion has arisen on the subject. Medical witnesses have often lost sight of two important points on which a correct answer to this inquiry must be based,—1, the law of the diffusion of gases; and 2, the effect of heat in greatly diminishing the specific gravity of a gas naturally heavy. There is no doubt that, in a narrow or confined vessel, exposed to air, carbonic acid is slow in escaping,—nevertheless it mixes and passes off with the air;—and in the course of an hour or two, in spite of its great specific gravity, none will be contained within the vessel. The well-known Grotto del Cane, at Pozzuoli, has been quoted by those who hold that carbonic acid always tends to remain on the lowest level; but it has been forgotten that in this, and other similar cases, carbonic acid is continually issuing from crevices in the soil, so that that which is lost by diffusion is continually replaced; hence the illustration proves nothing. It may suffice to state, that air and carbonic acid mix readily on contact in all proportions, although they enter into no chemical combination. Thus, then, at common temperatures, carbonic acid has no tendency to remain on the floor or soil, when there is a free access of air or contact with other gases. The heat of combustion diminishes the specific gravity of the gas, and the carbonic acid therefore ascends with the heated current of air, and diffuses itself in the upper part of an apartment, when there are no means of carrying it off. This is a fact demonstrable by many simple experiments. In burning a quantity of charcoal actively in an open brazier raised above the floor in a large apartment, I found that the proportion of carbonic acid was nearly equal in air taken from a foot above and a foot below the level of the source of combustion, there being no currents to affect the results. Hence it follows that carbonic acid produced by combustion has no particular tendency to collect at the lowest level; that it is uniformly diffused around, and probably it would be found by careful experiments, that within apartments of small dimensions—those in which individuals are often accidentally suffocated—the upper strata of air contain as much or even more carbonic acid than the lower. For this reason, a room with a low ceiling is more dangerous under these circumstances, than one which is high-pitched.

In a very large apartment, it would of course be improper to test the suffocating properties of the air, by the examination of it at a great distance from the source of combustion; since a person situated near this spot might be destroyed, while one at a distance might escape—the carbonic acid not having completely diffused itself; or supposing it to have become entirely diffused, the proportion may be so small as to render it harmless. It is well known, by the effects of the vapour of a lime-kiln, that one lying at the edge of the kiln may be destroyed, while another at ten yards distance, either on the same level or below it, may entirely escape; it would not be possible, in such a case, to speculate upon the proportion of carbonic acid which had here destroyed life, except by collecting the air from the spot where the accident occurred, and at or about the time of its occurrence. Another fallacy appears to be, that because a dead body is found recumbent, it is to be inferred that the individual must have lain down and have been destroyed while sleeping. The body of a dead person must always be found thus lying on a floor, unless it be supported; but suffocation may have actually taken place, or at least have commenced, when the deceased was in the sitting or erect posture. Admitting that carbonic acid diffuses itself rapidly from combustion in a small and closed apartment, it has been supposed that after having become mixed with the air, it would again in great part separate, and, owing to its superior density, fall to the lowest level on cooling. In answer to this it may be said—1. That there are no facts to support the opinion, while there are many against it; for we do not find that the heaviest and lightest gases, when once really mixed, ever again separate from each other. 2. Practically this explanation amounts to nothing; because before the gas had cooled and reacquired its density, its asphyxiating properties would probably have had their full effect on all living persons within its reach. Persons are not suffocated by carbonic acid after the fuel is extinguished, and the apartment cooled; but the poisonous action of the gas is commonly manifested while the fuel is still burning. The inferences which, it appears to me, we are entitled to draw from the preceding considerations, are—1. That in a small and close apartment, individuals are equally liable to be suffocated at all levels, from the rapid diffusion of carbonic acid during combustion. 2. That in a large apartment, unless the gas be very speedily diffused by a current of air,—the air around the source of combustion may become impregnated with a poisonous proportion, while that at a distance may be still capable of supporting life; because carbonic acid requires time for its perfect and equable diffusion in a very large space.

COAL-GAS. CARBURETTED HYDROGEN. CARBONIC OXIDE.

Since the introduction of coal-gas for the purpose of illumination, many fatal accidents have occurred from the respiration of air contaminated with it. Coal-gas is a very compound body, acting as a direct poison when respired. Its composition is subject to much variation, according to circumstances. Mitscherlich found that it was principally composed of light carburetted hydrogen, hydrogen, and carbonic oxide, in the proportion of 56 per cent. of the first, 21·3 of the second, and 11 of the third. M. Tourdes found that the proportions of light carburetted hydrogen and carbonic oxide were nearly equal, *i. e.* about 22 per cent.

This difference in composition depends on the heat to which the gas has been submitted. Some consider that CARBONIC OXIDE is the poisonous principle; but there is no doubt that the hydrocarbons also have a noxious influence, although the use of the safety-lamp in mines, proves that a mixture of protocarburetted hydrogen with air in a small proportion, may be respired without producing serious effects. The *symptoms* produced by coal-gas when mixed in a large proportion with air, are vertigo, cephalalgia, nausea with vomiting, confusion of intellect with loss of consciousness, general weakness and depression, partial paralysis, convulsions, and the usual phenomena of asphyxia. *Post-mortem appearances*:—These will be best understood from the following cases. In January 1841, a family residing at Strasburgh respired for forty hours an atmosphere contaminated with coal-gas, which had escaped from a pipe passing near the cellar of the house where they lodged. On the discovery of the accident, four of the family were found dead. The father and mother still breathed, but, in spite of treatment, the father died in twenty-four hours; the mother recovered. On a post-mortem examination of the five bodies, there was a great difference in the appearances; but the principal points observed were congestion of the brain and its membranes, the pia mater gorged with blood,—and the whole surface of the brain intensely red. In three of the cases, there was an effusion of coagulated blood on the dura mater of the spinal canal. The lining membrane of the air-passages was strongly injected; and there was spread over it a thick viscid froth tinged with blood; the substance of the lungs was of a bright red colour, and the blood was coagulated. (Ann. d'Hyg. Jan. 1842.) In two cases communicated by Mr. Teale to the Guy's Hospital Reports, (No. viii.) there was found congestion of the brain and its membranes, with injection of the lining membrane of the air-passages. In these cases, the blood was remarkably liquid. The circumstances under which the accident occurred, were very similar. An old lady and her grand-daughter, who had been annoyed by the escape of gas during the day, retired to bed, and were found dead about twelve hours afterwards.

In the cases above given, the effects produced by coal-gas were owing to its long continued respiration in a diluted state. The quantity contained in the air of the rooms must have been very small;—in M. Tourdes' case, it was probably not more than 8 or 9 per cent., because a little above this proportion the mixture with air becomes explosive, and there had been no explosion in this case, although in the apartment in which the individuals were found dead, a stove had been for a long time in active combustion, and a candle had been completely burnt out. In Mr. Teale's case, those who entered the house perceived a strong smell of coal-gas; but still the air could be breathed. Coal-gas, therefore, like all other aërial poisons, may destroy life if long respired, although so diluted as not to produce any serious effects in the first instance! This gas owes its peculiar odour to the vapour of naphtha:—the odour begins to be perceptible in air when the gas forms only the 1000th part;—it is easily perceived when forming the 700th part, but the odour is well marked when it forms the 150th part (Tourdes.) In most houses where gas is burnt, the odour is plainly perceived; and it is a serious question whether health and life may not be often affected by the long-continued respiration of an atmosphere containing but a small proportion. The odour will always convey a sufficient warning against its poisonous effects. It should be known that this gas will penetrate into dwellings in a very insidious manner. In Mr. Teale's case, the pipe from which the gas had escaped, was situated about ten feet from the wall of the bed-room where the female slept. The gas had permeated through the loose earth and rubbish, and entered the apartment through the floor! It is impossible to determine exactly what proportion of this gas in air will destroy life. An atmosphere containing from 7 to 12 per cent. has been found to destroy rabbits and dogs in a few minutes,—when the proportion was from 1½ to 2 per cent. it had little or no effect. With respect to man, it may destroy life if long respired when forming about 9 per cent., *i. e.* when it is in less than an explosive proportion. (See Brit. and For. Med. Rev. xxix. 253; also, Ann. d'Hyg. 1830, i. 457.)

M. Tourdes has ascertained that rabbits died in twenty-three minutes when kept in an atmosphere containing 1-15th of its bulk of pure *carbonic oxide.* When the proportion was 1-30th, they died in thirty-seven minutes, and when 1-8th, in seven minutes. Its action on the body is that of a pure narcotic.

ANALYSIS.—The circumstances under which the accident occurs, will generally suffice to establish the nature of the gas. Coal-gas burns with a bright white light, producing carbonic acid and water. A taper should be cautiously applied to a small quantity; since when the gas is mixed with air in the proportion of 11 to 14 per cent., it is dangerously explosive. For this reason no lighted candle should be taken into an apartment where an accident has occurred, until all the doors and windows have been for some time kept open. The combustion of the gas, or its explosion with air, is a sufficient test of its nature;—the peculiar odour and the want of action on a salt of lead, will distinguish it from sulphuretted hydrogen.

Carbonic oxide is known by its burning with a pale blue light, and producing carbonic acid and water by its combustion.

SULPHURETTED HYDROGEN. AIR OF DRAINS AND SEWERS.

General remarks.—This gas in a toxicological point of view, may be considered next in importance to carbonic acid. Individuals are occasionally accidentally killed by it; but the very offensive odour which a small portion of it communicates to a large quantity of air, is sufficient to announce its presence, and to prevent any dangerous consequences from taking place. The sulphuretted hydrogen gas, when respired in its pure state, is almost instantaneously mortal. It exerts equally deleterious effects upon all orders of animals, and upon all the textures of the body. It is found to destroy life, even when it is allowed to remain in contact with the skin. Mr. Donovan states that a rabbit enclosed in a bladder of sulphuretted hydrogen gas, but allowed to breathe freely in the atmosphere, perished in ten minutes. When introduced into the lungs of animals, even in a very diluted state, it has been known to give rise to fatal consequences. Thus, Thénard found that air which contained only one eight-hundredth of its volume of this gas, would destroy a dog; and that when the gas existed in the proportion of one two-hundred-and fiftieth, it sufficed to kill a horse. The later researches of M. Parent-Duchâtelet, however, seem to show that the poisonous effects of the gas have been somewhat exaggerated, at least in the application of these results to man. He observed that workmen breathed with impunity, an atmosphere containing one per cent. of sulphuretted hydrogen, and he states that he himself respired, without serious symptoms ensuing, air which contained *three per cent.* In most drains and sewers, rats and other vermin are found to live in large numbers; and according to Gaultier de Claubry, the air in these localities contains from two to eight per cent. (Devergie, ii. 520.) Thus, admitting it to be a poison even more powerful than carbonic acid, it does not appear to be energetic as Thénard's experiments would lead us to suppose. An atmosphere containing from six to eight per cent. of the gas, might speedily kill, although nothing certain is known of the proportion required to destroy human life. One fact, however, is worthy of the attention of medical jurists, namely, that the respiration of an atmosphere, only slightly impregnated with gas, may, if long continued, seriously affect an individual, and even cause death. M. d'Arcet had to examine a lodging in Paris, in which three young and vigorous men had died successively, in the course of a few years, under similar symptoms. The lodging consisted of a bed-room with a chimney, and an ill-ventilated ante-room. The pipe of a privy passed down one angle of the room by the head of the bed, and the wall in this part was damp from infiltration. At the time of the examination there was no perceptible smell in the room, although it was small and low. M. d'Arcet attributed the mortality in the lodging to the slow and long-continued action of the emanations from the pipe; and it is highly probable that this was the real cause. (Ann. d'Hyg. Juillet 1836.) The men who were engaged in working at the Thames Tunnel, suffered severely during the excavation from the presence of this gas in the atmosphere in which they were obliged to work. The case was referred to me for examination by Sir M. I. Brunel, 1839. The air as well as the water was found to contain sulphuretted hydrogen, which trickled through the roof. It was probably derived from the action of the water on the iron-pyrites in the clay. The gas issued in sudden bursts, so as to be at times perceptible by its odour. By respiring this atmosphere, the strongest and most robust men, were in the course of a few months reduced to an extreme state of exhaustion, and several died. The symptoms with which they were first affected, were giddiness, sickness and general debility; they became emaciated and fell into a state of low fever, accompanied by delirium. In one case which I saw, the face of the man was pale, the lips of a violet hue, the eyes sunk with dark areolæ around them, and the whole muscular system flabby and emaciated. Chloride of lime and other remedies were tried for the purification of the air; but the evil did not entirely cease until the tunnel was so far completed that there was a communication from one side to the other, and free ventilation throughout.

SYMPTOMS.—The symptoms produced by sulphuretted hydrogen on the human system, vary according to the degree of concentration in which it is respired. When breathed in a moderately diluted state, the person speedily falls inanimate. An immediate removal to pure air, venesection, and the application of stimulants, with cold affusion, may, however, suffice to restore life. According to the account given by those who have recovered, this state of inanimation is preceded by a sense of weight in the epigastrium and in the region of the temples, also by giddiness, nausea, sudden weakness and loss of motion and sensation. If the gas in a still less concentrated state, be respired for some time, coma or tetanus with delirium supervenes, preceded by convulsions or pain and weakness over the whole of the body. The skin, in such cases, is commonly cold, the pulse irregular and the respiration laborious. When the air is but very slightly contaminated by the gas, it may be breathed for a long time without producing any serious symptoms; sometimes there is a feeling of nausea or sickness, accompanied by pain in the head, or diffused pains in the abdomen. These symptoms are often observed to affect those who are engaged in chemical manipulations with this gas. Sulphuretted hydrogen appears to act like a narcotic poison when highly concentrated; but like a narcotico-irritant when much diluted with air. It is absorbed into the blood, to which it gives a browish black colour, and it is in this state circulated throughout the body.

POST-MORTEM APPEARANCES.—On examining the bodies of persons who have died from the effects of sulphuretted hydrogen, the following appearances have been observed. The mucous membrane of the nose and fauces is commonly covered by a brownish viscid fluid. A highly offensive odour is exhaled from all the cavities and soft parts of the body. These exhalations, if received into the lungs of those engaged in making the inspection, sometimes give rise to very unpleasant symptoms, and may even cause syncope or asphyxia. The muscles of the body are of a dark colour, and are not susceptible of the galvanic stimulus. The lungs, liver, and the organs generally, are distended by black liquid blood. There is also great congestion about the right side of the heart, and the blood is said not to become coagulated after death: the body rapidly undergoes the putrefactive process.

Drains and Sewers.—The most common form of accidental poisoning by sulphuretted hydrogen, for it is rare that a case occurs which is not purely accidental, is witnessed in nightmen and others who are engaged in cleaning out drains and sewers, or in the removal of the soil of privies. These accidents are much more frequent in France than in England, the soil being often allowed to collect in such quantities in Paris and other large continental cities, as to render the removal of it, a highly dangerous occupation for the workmen. According to the results of Thénard's observations, there are two species of compound gases or mechanical mixtures of gases, which are commonly met with in the exhalations of privies. The first compound consists of a large proportion of atmospheric air holding diffused through it, in the form of vapour, the *Hydrosulphuret of ammonia*. The hydrosulphuret is contained abundantly in the water of the soil, and is constantly rising from it in vapour, and diffusing itself in the surrounding atmosphere. It is this vapour which gives the highly unpleasant odour, and causes an increased secretion of tears in those who unguardedly expose themselves to such exhalations. The *symptoms* produced by the respiration of this gaseous mixture when in a concentrated state, bear a close resemblance to those which result from the action of sulphuretted hydrogen gas. If the person be but slightly affected, he will probably complain of nausea and sickness, his skin will be cold, his respiration free but irregular; the pulse is commonly frequent, and there are spasmodic twitchings of the voluntary muscles, especially of those of the chest. If more seriously affected, he loses all power of sense and motion, the cutaneous surface becomes cold, the lips and face assume a violet hue, the mouth is covered by a sanguineous mucous, the pulse is small, frequent, and irregular; the respiration hurried, laborious, and convulsive; and the limbs and trunk are in a state of general relaxation. In a still more severe degree, death may take place immediately; or should the person survive a few hours, in addition to the above symptoms, there will be short but violent spasmodic twitchings of the muscles, sometimes even accompanied by opisthotonos. (See Ann. d'Hyg. 1829, ii. 70.) If the individual be sensible, he will commonly suffer the most severe pain, and the pulse may become so quick and irregular that it cannot be counted. When the symptoms are of such a formidable nature, it is very rare that a recovery takes place. The *appearances* met with on making a post-mortem examination of the body, are similar to those observed in death from sulphuretted hydrogen. The inspection should be made with caution, for a too frequent respiration of the poisonous exhalations may seriously affect the practitioner. The *treatment* is the same as in poisoning by carbonic acid.

A singular accident occurred in this metropolis in August 1847, in which a man lost his life by the evolution of a quantity of sulphuretted hydrogen from a foul drain. It appears that shortly before the accident, a large quantity of oil of vitriol had been poured down the drain communicating with a privy. The deceased entered the yard, and was soon afterwards found on the pavement in a dying state. On inspection of the body, the brain was healthy; but the lungs were gorged with blood, which had the offensive odour of the sulphuretted hydrogen gas. The medical witness referred death to this gas, and stated that lime had been thrown into the drain, that sulphuret of calcium had probably been formed, and that the sulphuretted hydrogen, which had led to the death of the deceased, had been evolved from this by the vitriol. It is more probable, however, that the gas was evolved by the decomposition of the hydrosulphuret of ammonia, which always abounds in such localities.

Analysis.—The recognition of these gases is a very simple operation. The odour which they possess is sufficient to determine their presence, even when they are diluted with a large quantity of atmospheric air. The *sulphuretted hydrogen gas* is at once identified by its action on paper previously dipped in a soluble salt of lead: if present even in very small proportion, the moistened paper speedily acquires a brownish black stain from sulphuret of lead. The sulphuretted hydrogen may be also thus proved to exist in the vapour of *hydrosulphuret of ammonia* mixed with air; and the presence of ammonia is indicated in the compound, by the volatile alkaline reaction on test-paper, also by holding in the vessel containing the vapour recently collected, a rod dipped in strong muriatic acid: the production of dense white fumes announces the formation of muriate of ammonia. It is a fact which cannot be too universally known, that a candle will readily burn in a mixture of either of these bodies with air, which, if respired, would suffice to destroy life. (Ann. d'Hyg. 1829, ii. 69.) It is also worthy of remark, that the air of a cesspool may be often respired with safety until the workmen commence removing the soil, when a large quantity of mephitic vapour may suddenly escape, which will lead to the immediate suffocation of all present. Several persons have been killed by trusting to the burning of a candle, in ignorance of this fact. The best plan for getting rid of the gas is by a free exposure of the locality, or by exciting active combustion in it. According to Parent Duchâtelet, men can work in an atmosphere containing from two to three per cent. of sulphuretted hydrogen. The air of one of the principal sewers of Paris gave the following results on analysis in 100 parts: oxygen, 13·79; nitrogen, 81·21; carbonic acid, 2·01; sulphuretted hydrogen, 2·99.

There is another species of deleterious compound present in these exhalations of a very different nature. It is more rarely met with than the preceding, and consists, according to Thénard, in 100 parts, of nitrogen 90, of oxygen 2, and carbonic acid 4. Sometimes the carbonic acid gas is combined with ammonia, and then it may be regarded, chiefly, as a mixture of nitrogen holding diffused through it the vapour of carbonate of ammonia, which is sufficient to render it highly irritating to the mucous membrane of the eyes and nose. Its action on the human body when respired, will be readily understood from this statement of its chemical composition. In its operation, it must be regarded as exerting an influence essentially negative; for the small proportion of carbonic acid, or of carbonate of ammonia existing in it, cannot be supposed to give rise to the asphyxia which so rapidly follows its inhalation. The chances of recovery are much greater in persons who become asphyxiated from the inspiration of this compound, than in those who are exposed to the influence of the preceding. Commonly the immediate removal to a pure air is sufficient to bring about a recovery; for the asphyxia is originally induced, owing to there being an insufficient portion of oxygen in the mixture to sustain life. Should death take place, it will be found on a post-mortem inspection, that the internal appearances are the same as those which are met with in the examination of the bodies of the hanged or the drowned.

Analysis.—This compound extinguishes a taper: the carbonic acid contained in it may be removed by caustic potash, and then it will be seen that the great bulk of the mixture is formed of nitrogen,—a gas which, by its negative properties, cannot be easily confounded with any other. In a mixed atmosphere of carbonic acid and sulphuretted hydrogen, the two gasses may be separated by agitating the mixture with a solution of acetate of lead, and treating the precipitate with acetic acid, which dissolves the carbonate, and leaves sulphuret of lead.

EXHALATIONS OF THE DEAD.

It may not be inappropriate to make a few remarks in this place, on the alleged danger of the exhalations given off by dead bodies in a state of putrefactive decomposition. Formerly there existed a groundless fear relative to the examination of a putrified dead body; and during the last century, on several important occasions, medical witnesses refused to examine the bodies of deceased persons, who were presumed to have been murdered, alleging that it was an occupation which might be attended with serious consequences to themselves. Orfila has collected many accounts of the fatal effects which are recorded to have followed the removal of the dead some time after interment. (Traité des Exhumations, vol. i. p. 2, et seq.) He allows, however, that the details of most of these cases are exaggerated, and attributes the effects which followed to other causes. Indeed, the observations of Thouret and Fourcroy prove that these dangers are restricted within a very narrow compass, and that in general with common precautions, the dead may be disinterred and transported from one locality to another, without any risk to those engaged in carrying on the exhumations. About the latter part of the last century, from fifteen to twenty thousand bodies, in almost every stage of decomposition, were removed from the Cimetière des Innocens in Paris; and the accidents that occurred during the operations, which lasted ten months, were, comparatively speaking, few. The workmen acknowledged to Fourcroy, that it was only in removing the recently interred corpses, and those which were not far advanced in decomposition, that they incurred any danger. In these cases, the abdomen appeared to be much distended with gaseous matter,—if ruptured, the rupture commonly took place about the navel, and there issued a sanious fetid liquid, accompanied by the evolution of a mephitic vapour, probably a mixture of carbonic acid and sulphuretted hydrogen. Those who respired this vapour at the moment of its extrication, fell instantly into a state of asphyxia and died; while others, who were at a distance, and who consequently respired it in a diluted state, were affected with nausea, vertigo, or syncope, lasting for some hours, and followed by weakness and trembling of the limbs. Chloride of lime was formerly employed for decomposing these vapours; but a strong solution of nitrate of lead, or chloride of zinc, has been lately proposed as a less offensive preparation.

Several lives have been lost of late years from the crowded state of the burial-grounds of London. A deep grave is dug, and this is kept open to be piled with coffins until filled. Persons venturing into these graves are immediately suffocated. The earth in these localities is strongly impregnated with noxious exhalations; and no excavation can be made without its becoming immediately converted into a well of carbonic acid! This appears to be the poisonous gas to which fatal accidents in these localities are most commonly due. (See on this subject Henke's Zeitschrift, 1840, ii. 446. Ann. d'Hyg. 1832, 216; 1840, 131; 1843, 28, 32.)

In addition to these there are other gases of a poisonous nature which are for the most part artificial products. It is seldom that individuals are exposed to respire them in such quantity as to cause serious symptoms or endanger life: hence they require but a brief notice in this place.

CHLORINE.

The irritant properties of chlorine when dissolved in water, have been elsewhere described. (Page 246, ante.) In a concentrated state, the attempt to respire this gas would probably be attended with the immediate destruction of life. When diluted, even with a large proportion of air, it has a peculiar and pungent odour; it is highly irritating if respired,—exciting cough, and in some instances spitting of blood. It is said that in chemical manufactories, the workmen become habituated to this gas, and breathe it in a moderately diluted state without inconvenience. On the other hand, instances are met with in which individuals appear to be peculiarly susceptible of its noxious effects. When it does not immediately destroy life, it may give rise to inflammation of the air-passages and lungs, and thus slowly lead to death. The celebrated chemist Pelletier is said to have lost his life from this cause. *Treatment.*—Removal to a free current of air; the inhalation of the diluted vapour of ammonia. *Analysis.*—Its peculiar pungent odour, and its green colour, are sufficient to identify chlorine. It bleaches organic colouring matters when in a humid state; and a lighted taper burns in it with a red smoky flame.

MURIATIC (HYDROCHLORIC) ACID GAS.

This gas is copiously liberated in the manufacture of soda from sea-salt. It is quite irrespirable when in a concentrated state, and produces all the effects of a powerful local irritant on the lungs and air-passages when breathed in a diluted state. Animals soon perish if kept in an atmosphere impregnated with only a small quantity of the gas. I am not aware of any instance in which it has proved fatal to man. The air of a locality infected with it in any degree perceptible to the senses, would undoubtedly be injurious to health, and might give rise to chronic disease of the lungs. Certain questions have frequently been raised in Courts of Law respecting the influence of this gas on vegetables; and actions for nuisances on this ground have been frequently brought against the owners of Soda-works. This subject has been investigated by Drs. Christison and Turner, and the Messrs. Rogerson. All agree in representing muriatic acid gas as exerting a most pernicious influence on vegetable life. Thus it was found by the last-mentioned experimentalists to be injurious in direct proportion to the quantity, even up to a dilution with 1500 parts of air. The tips and edges of the leaves of trees and plants, as well as the ends and branches with the young shoots, are first destroyed; and even where it does not reach a 1500th part, and does not visibly produce these effects, it still impairs the growth of the plant. (Med. Gaz. x. 315.)

Treatment.—If respired,—the diluted vapour of ammonia. *Analysis.*—The absorption of the gas by distilled water, and the production of a white flocculent precipitate of chloride of silver on the addition of a solution of nitrate of silver.

COMPOUNDS OF NITROGEN.

NITROGEN itself is not poisonous (ante, page 636.) It sometimes abounds in the air of drains, but always mixed more or less with other gases (page 651, ante.) The two gaseous compounds of nitrogen and oxygen exert a poisonous action on the body.

PROTOXIDE.—This, which is called the Laughing gas, is well known by its producing a species of intoxication, with great excitement and exhalation of the muscular powers. It has a sweet taste, but is quite inodorous. During the respiration of this gas, the face becomes pallid: the lips assume a violet hue, indicative of its effect on the blood; and a series of pleasurable sensations are said to pass through the mind with inconceivable rapidity. These singular effects were discovered by Sir H. Davy in experiments made upon himself. In one instance mentioned by Professor Silliman, the after-effect of the gas was a complete perversion of the sense of taste. The subject of this experiment could only take articles of food which were saccharine: thus for several days he lived upon sweet cake. For more than eight weeks after inspiring the gas, he was in the habit of eating beef, fish, poultry, potatoes, and all kinds of food mixed with a large quantity of treacle or molasses! This gas operates by over-stimulating the system. It speedily causes the death of an animal, and would destroy a human being if long respired. Some serious after-effects on the lungs and brain, have occasionally followed the inhalation of it in common experiments.

Analysis.—An extinguished taper with an ignited wick is rekindled in this gas, as it is in oxygen. The protoxide differs from oxygen, in not forming red acid fumes when mixed with the deutoxide of nitrogen.

Deutoxide of Nitrogen.—Sir H. Davy suffered severely by an attempt to respire this gas. It produces, when mixed with air, red acid (nitrous acid) vapours, which are highly irritant and corrosive. Severe inflammation of the fauces and lungs would probably follow any attempt to breathe it in a pure state, even if the individual recovered from the first effects. An alleged fatal case of poisoning by this gas is reported by Dr. Mans in the American Journal of Med. Sciences, Oct. 1846, 380. The man is stated to have respired the fumes of nitrous acid in a Sulphuric Acid factory. While breathing the vapour he complained of no pain, and only felt unwell some hours afterwards. With the exception of a hacking cough, the symptoms had none of the well-marked characters of this form of gaseous poisoning. He died the next morning, and on inspection, the lungs were found in a diseased condition, and there were strongly developed signs of gastritis. It is on the whole very doubtful whether the death of this person could be ascribed to the effects of the nitrous acid vapour.

NITROUS ACID.

It has been already remarked that the Deutoxide of Nitrogen, or Nitrous Gas, cannot be respired in air except under the form of *Nitrous acid vapour*. Workmen who are employed in gilding in the humid way are exposed to these fumes, and occasionally suffer severely from them. This subject has been recently investigated by MM. Chevallier and Boys de Loury. (Ann. d'Hyg. Oct. 1847, 323.) It appears that in the Parisian workshops, the fumes, respired to only a small extent, and probably much diluted, do not materially affect the health of the men; but the following cases will show that fatal effects may ensue when they are breathed in a concentrated state. A man who dealt in Aqua Fortis accidentally breathed the acid fumes: he felt at the time as if he should be suffocated. In about two hours he perceived a dry and burning heat in the throat, accompanied by irritation of the stomach and chest, and a sense of constriction in the epigastrium. He passed several motions of a light yellow colour: there was a great desire to micturate, but no urine was passed. In the course of the day he expectorated matter of a yellowish colour. Enemata were employed, and these brought away yellow-coloured evacuations. The symptoms became aggravated towards evening; there was mucous rattle, hiccough, severe pain in the region of the diaphragm, convulsive motions, and slight delirium. Death took place in about twenty-seven hours after the respiration of the acid fumes. The abdomen became much swollen after death. A dog which had been exposed to the fumes died in about two hours.

A young man, while pouring a large quantity of nitric acid into a metallic vessel, respired the fumes, which at the time produced a most violent cough. He felt uneasy during the day, but it was not until the evening that the symptoms became alarming. Respiration was difficult, and could be performed only while the patient was in the sitting posture. He suffered from a frequent dry cough, which, after some effort, led to the expectoration of an orange-yellow coloured frothy matter: the pulse was hard and full, and some blood which had been abstracted was of a very dark colour: he became speechless, and died in two days. On inspection, the whole of the right lung was found disorganized, and gorged with black liquid blood;—the left presented the same appearance, but in a less degree: the heart was gorged with dark-coloured blood: the lining membrane of the trachea and bronchi was of a livid colour, and the uvula and mucous membrane of the fauces were in a gangrenous condition. The stomach was enormously distended by gases, so acid as to corrode the silver of the instruments used in the inspection; the mucous membrane was thickened, and towards the cardia destroyed. The intestines were distended with gas, and their coats were of a pale red colour.

In June 1844, Dr. Sucquet was called to a man who had been occupied in a small closet in cleaning copper cases with diluted nitric acid. He suffered at first from cough, pain in the head, and general oppression. When seen by Dr. Sucquet the following morning, his eyes were prominent, his lips of a violet colour, and his speech short and interrupted. He coughed frequently, expectorated a large quantity of a yellow-coloured thready liquid, and his breathing was short and painful. The skin was cold; pulse 98, small and regular; urine not freely passed. He died in twenty-eight hours after the attack. On inspection, the mucous membrane of the lungs was found thickened, and more red than natural: the bronchia contained a great quantity of a yellowish-coloured liquid: the brain was injected. In all these cases the intellect was uniformly preserved until the last. Three other instances are mentioned, in which the patients recovered: the leading symptoms were dyspnœa, cough, and burning pain in the stomach. It will be perceived that they differ from those described in the case of Dr. Mans.

Analysis.—The production of the red fumes of nitrous acid on exposure to air is sufficient to identify the deutoxide of nitrogen, while nitrous acid is known by its odour, its orange-red colour, acid reaction and entire solubility in water.

The other gases require no particular notice. Oxygen, according to the experiments of Mr. Broughton, acts, when long respired, as a narcotic poison to animals. It is a question, however, whether the carbonic acid evolved during the respiration of it, was not really the narcotic agent. The atmosphere which we daily breathe contains *one-fifth* of its volume of this gas in a *free* state; and however long respired, no narcotic effects, even in the smallest degree, are produced by it. Can the admixture of this gas with four times its bulk of another gas, destitute of any positive action, entirely neutralize its narcotic properties? This is so improbable that it appears more reasonable to consider that oxygen is not a narcotic poison. It most probably destroys life by over-stimulating the nervous system. Cyanogen is a highly poisonous gas, and could not be breathed, even in a diluted state, without great risk of life. It produces in animals coma, convulsions, and death. Bromine and Iodine in vapour, and the Hydrobromic and Hydriodic acid gases, may be regarded as corrosive and irritant poison, highly dangerous to respire under any circumstances.

APPENDIX.

Page 18. *The effects produced by cold liquids.*—The effects produced by cold liquids, sometimes rather closely resemble those caused by poison. A girl, æt. 9, during sultry weather, and while in a heated state, drank a coffee-cupful of cold water, She immediately fell to the ground in a state of insensibility. When seen by a medical man half an hour afterwards, she was quite unconscious: the skin was cold, the pulse feeble, and the pupils were unaffected by light. There were also convulsive twitchings about the corners of the mouth. She was bled, stimulants were applied, and in about five or six hours she recovered. (Lancet, Oct. 7, 1843.)

Page 20. *Is liquid mercury a poison?*—The facts mentioned at page 310, appear to me to show clearly that metallic mercury cannot be regarded as a noxious or deleterious substance. Orfila states that it may become poisonous when it remains so long in the body as to undergo oxidation. (Toxicologie, i. 600.) He quotes one case in which the mercury from being retained for a period of fourteen days, excited abundant salivation, with ulceration of the mouth and paralysis. This, however, must be considered as a rare example; and it proves that the oxide, rather than the metal, is injurious. Mr. Nicol has suggested that mercury given in a finely divided form, as in the state in which it is precipitated from its solution by chloride of tin, may have an energetic action. (Brit. Amer. Jour., Aug. 1847.) This is possible, as the metal is brought into a condition better fitted for absorption. The action of mercury with chalk, and some other mercurial preparations, may probably be dependent on a mixture of the *metal* in a fine state of division with oxide of mercury.

Page 23. *Mechanical irritants.*—A very singular case is reported in the Dublin Medical Press (Aug. 19, 1846, p. 117), in which a female, æt. 19, an imbecile from childhood, had acquired the practice of swallowing portions of her own *hair*. The symptoms shortly before her death were, great emaciation, pulse small and weak, constant vomiting after taking food, which was always liquid, as the slightest solid caused excessive pain. There was a hard tumour perceptible in the epigastrium. The real cause of death was not even suspected, until an inspection was made, when it was found that the stomach was distended by a large mass of human hair.

The effects of mechanical irritants are further shown by the case of a child, who suffered from diarrhœa, heat in the epigastrium, coldness of the extremities, and other symptoms, in consequence of her having swallowed some copper *percussion-caps*. The alvine irritation disappeared after twenty-four hours; the caps were discharged, and the child recovered. (Philadelphia Med. Examiner, June 1847, 332.) A very singular case is quoted by Dr. Frank, in which a child, æt. three years, died from the effects of the *tooth* of a viper (*Trigonocephalus,*) which it had swallowed. The mucous membrane of the stomach was of a cherry-red colour, and the foreign body, covered by a layer of sloughy membrane, was found near the pylorus. (Casper's Wochenschrift, Aug. 14, 1847, 531.)

Page 30. *On the absorption and elimination of poisons.*—In the text I have not adverted to the very interesting experiments on absorption and elimination performed by Mr. Erichsen, in a case of extroversion of the bladder; because these experiments were performed with substances not ranked among poisons. Nevertheless, as the results are calculated to throw some light upon the differences in the rate of absorption under different states of the body, it will be proper to call the reader's attention to them. The ferrocyanide of potassium, swallowed in solution, was detected in the urine at different periods,—the earliest period being only *one minute* after it had been swallowed, and the longest *thirty-nine minutes.* An analysis of the cases showed that the time of its appearance depended, in this secretion, upon the state of the stomach when the salt was swallowed: the earliest periods being when the stomach was empty, and the latest when it was full, and the process of digestion going on. Some vegetable salts and infusions were tried, and the only inference deducible was, that they required a much greater average time for their passage through the system, than the ferrocyanide of potassium. They gave some indications in the urine in from sixteen to thirty-five minutes.

As to *elimination* by the urine, it was found that when the dose of ferrocyanide of potassium was forty grains, and its presence in the urine was manifested in two minutes, no trace of it could be detected after the lapse of twenty-four hours. Supposing the whole of it to have been eliminated by the urine, it must have escaped at an average rate of a grain in thirty-six seconds. It is rather remarkable that, when the dose was reduced by one-half, the salt was detected in the urine for an equal, and in one instance for a longer period of time (twenty-eight hours.) Hence the rate of elimination, like that of absorption, is by no means uniform. (For a full account of Mr. Erichsen's ingenious experiments, I must refer the reader to the Medical Gazette, xxxvi. 363, 410.) The results give a fair analogical explanation of the fact why poisons taken on an empty stomach operate with such great rapidity. The effect of animal poisons may be similarly influenced: thus the poison introduced into the system by dissection-wounds, or by handling the viscera and liquids of the dead body, when there is abrasion on the fingers, may, if the stomach be empty, lead to its very rapid absorption and diffusion.

From the researches of MM. Millon and Laveran, it appears that there may be intermissions in the elimination of certain metallic poisons. In giving to their patients from one grain and a half to five grains of tartarized antimony, they remarked that it was eliminated by the urinary secretions, but in some instances slowly and unequally. They therefore examined the urine, not only several days after the introduction of the medicine, but for some days after it had ceased to appear in this secretion. They then found that its elimination underwent a well-marked intermission, and that, in a most unexpected way, it appeared to remain for a certain period fixed in the body. In two patients they detected traces of it twenty-four days after its administration. In the body of one who died of phthisis, they found antimony in the liver. In a third case, antimony was detected in the urine after twenty days; in two others, after nineteen days; and in three others after sixteen, seventeen, and eighteen days respectively. (Comptes Rendus, 1845, ii. 638.) The fact of the *intermittent elimination* of poisons is of importance, as it tends to show that, even when traces of poison cease to appear in the secretions, it does not in all cases follow that the substance is entirely expelled from the body.

Page 32. *On the transmission of poisons by absorption from the mother to the fœtus.*—M. Audouard ascertained, in his experiments on rabbits, that when mineral poisons were administered, so as not to produce death rapidly, distinct traces of the substance might be detected in the organs of the fœtus; and that, when death has taken place with great rapidity, indications of the presence of poison, may always be found in the placenta. A woman, during the last four months of her pregnancy, had been under treatment with iodide of potassium. The liquor amnii, collected during parturition, was clearly proved to contain iodine. (Comptes Rendus, 1845, i. 878.) This observation has been confirmed by the more recent experiments of M. Flandin. He found that by the use of mineral poisons, and especially of arsenic, he could induce abortion in rabbits; and the bodies of the young invariably yielded traces of the poison. He thinks also, that in poisoning the blood through absorption, abortion may be induced, by the destruction of the fœtus in the living female through the medium of the poisoned liquid. Abortion was thus caused in animals in three different experiments, by the agency of morphia,—in two female rabbits, which took the poison with their food, and in a bitch, which received the poison by subcutaneous absorption. (Gaz. Méd. 31 Juillet, 1847, 620.) This throws a new light upon the subject of abortion. It has been commonly supposed that the shock to the system, and not the poisoning of the fœtus, was the primary cause which led to the expulsion of the contents of the uterus. The observations receive corroboration from those made by Dr. Beattie, that the ergot of rye, given in large doses to females during parturition, may exert a poisonous action on the fœtus, and lead to its expulsion dead (ante, p. 434.)

Mr. Hicks has shown by an interesting experiment, that it requires a certain time in order that a poison should be transmitted by the blood from the mother to the fœtus. On one occasion he killed a pregnant cat by pouring a drachm of prussic acid down its throat. The chest was opened two minutes after death, and the heart was seen contracting. On laying open the abdomen and examining the uterus, the kittens were found alive, not having apparently suffered in the least from the poison, as they continued to move for some time even when removed from the uterine cavity. This observation appears to show that the parent cat did not die from the blood becoming poisoned by absorption; because if the blood were so poisoned as to kill the mother, it is difficult to understand how the kittens should have escaped. (Med. Gaz. xxxvi. 590.)

A case communicated to the same journal by Mr. Steele, renders it highly probable that the *milk* of the mother may become a medium for the transmission of poison. Two ewes were bitten by a rabid dog. Rabies appeared in them about six weeks after the bite, and they were killed. One had two lambs, the other one. At first they were permitted to suckle. The lambs were subsequently attacked with rabies, and were then killed. It appears highly probable that they received the poison through the milk, because they were removed from the ewes a month before these became affected: there was no mark of their having been bitten, nor is it proved that a sheep can communicate the poison by a bite, either before or after it has been attacked with rabies. (Med. Gaz. xxv. 160.)

Page 34. *Action of poisons independently of absorption.*—In the text it is stated that some poisons, of which prussic acid may be taken as an example, act so rapidly, that absorption cannot be necessary to their fatal effects. Several illustrative instances are given, and others will be found at page 529. As animals have died from this poison in *two* or *three seconds*, unless it be proved that the acid can be absorbed and circulated within this period of time, it is clear that in these cases at least, the poison must act in some other way:—absorption cannot be necessary to its fatal influence. If future experimentalists should succeed in showing that the process may be completed within less than two seconds, this argument would be untenable; but then there is the difficulty that in the poisoning of pregnant animals by prussic acid, the fœtus are not destroyed. (See the preceding paragraph.)

It has been contended that these instances of rapid death are not even exceptional cases; because when the poison is introduced by the mouth, the *vapour* penetrates into the lungs by a single inspiration, and thus the acid enters the blood, and produces its fatal effects. If this explanation were true, it is difficult to comprehend why death should not be almost instantaneous in every instance of poisoning by prussic acid; and why in its administration by the mouth, death should not be in every case much more rapid than when the acid is applied to the mucous membrane of the eye or rectum. Mr. Nunneley found, in his experiments, that the poison acted with almost if not absolutely equal rapidity when applied upon a mucous membrane, as the conjunctiva, rectum, or vagina, as when swallowed. (Prov. Trans. N. S. iii. 84.) In one experiment, No. 25, the dose by the eye produced much more decided effects than by the mouth. The experiments of Dr. Gerecke of Linz, are confirmatory of the view that death takes place too rapidly to admit of the supposition that absorption is, in *all* instances, necessary to the operation of this poison. (Casper's Wochenschrift, 26 September, 1846, 615.)

Page 54. *Diagnosis of cholera and arsenical poisoning. Case of the Duke of Praslin.*—In the text it is recommended as one method of diagnosis to make an analysis of the evacuated liquids. The omission of this duty led to a very erroneous opinion on the part of some eminent physicians in the case of the *Duke of Praslin.* This nobleman, in August 1847, having destroyed his wife, committed suicide by taking arsenic. The case involved many remarkable points in reference to this poison:—1, the distinction between its effects and the symptoms of common cholera; 2, the latest period at which well-marked symptoms appear after a large dose; 3, remission and apparent recovery with subsequent death; 4, death from a large dose after the period of six days.

The Duke was arrested early on the Wednesday morning; the symptoms did not appear until Wednesday evening. From this medical fact, as well as from moral circumstances, there can be no doubt that the poison was taken by the deceased shortly before their occurrence, and while he was under arrest. The symptoms which appeared *suddenly* on Wednesday evening were,—incessant vomiting and purging during the whole of the night and the following day, icy coldness of the extremities, thready pulse, and great prostration of strength. It is remarkable that there was an absence of pain: but this is one of the anomalous characters of arsenical poisoning, (see p. 259.) On the Friday, there was a remission in the symptoms of alvine irritation,—a condition not incompatible with irritant poisoning, (see p. 44;) but the cause of the symptoms would have been at once determined by an analysis,—as the matters vomited just before the remission, were found by a chemical examination subsequently made, to contain arsenic. In reference to the last point, it is a common error to suppose that a large dose of arsenic must necessarily destroy life rapidly (ante. p. 272.) A case occurred at Guy's Hospital in October 1847, in which a man did not die until *the seventh day,* although he had taken a teaspoonful and a half (220 grains) of arsenic.

Pages 78 and 81. *Oxide of Iron and Magnesia as antidotes to Arsenic.*—M. Caventou states, that the arsenites of iron and magnesia are rendered soluble and therefore poisonous by the muriate of ammonia, which is copiously secreted from the mucous membrane of the stomach. He has ascertained that the arsenites commonly regarded as insoluble are readily dissolved by this alkaline salt, which, as it is poured out, has often a strongly acid reaction. The relatively solvent power of a saturated solution of the muriate on the arsenites is indicated by the following numbers:—It required 115 parts of the muriate to dissolve arsenite of lime; 330 parts to dissolve arsenite of magnesia; and 600 parts to dissolve arsenite of iron. Hence, cæteris paribus, the arsenite of iron is not so readily dissolved as the other two arsenites. The dense or highly calcined magnesia of the shops, is less efficacious than the lighter varieties, in consequence of the greater cohesion of its particles. (Gaz. Méd. Sept. 11, 1847, 729.)

Page 88. *After-treatment of irritant poisoning.*—A judicious observation made by Dr. Corfe of the Middlesex Hospital, (Med. Times, Oct. 2, 1847, 629,) reminds me that I have not laid sufficient stress upon one part of the after-treatment, when an individual is recovering from irritant poisoning. As recovery is almost always attended with an inflammatory condition of the alimentary canal, —often increased by too frequent administration of irritant emetics, such as sulphate of zinc, the patient should not be allowed to overload the stomach with liquid or solid food. Like an inflamed eye, the organ requires repose, in order that its functions should be re-established. If there be intense thirst after the danger from the primary effects of the poison is over, a small quantity of barley-water or some bland mucilaginous liquid should be given occasionally. It is better that the thirst should be borne, than that an inflamed or an irritated organ should be distended with a pint or a quart of fluid at intervals of a few minutes.

Page 162. *Immediate occurrence of symptoms in poisoning by sulphuric acid.*—In a case tried at the York Assizes in July 1846, a man was convicted of the murder of his infant child by the administration of oil of vitriol. The moral circumstances showed that the poison had been procured by the prisoner; that the infant was not likely to have taken it itself; and although the child had been frequently fed during the evening, the commencement of the action of the poison was traced to a few minutes during which the child was left alone in the care of the prisoner. Here the medical evidence had a very important bearing, for it was most properly insisted that the effects caused by oil of vitriol are almost instantaneous, and admit of no delay in their production. The prisoner's defence was, that he was quite innocent of the charge, and that he had neither bottle, nor spoon, nor any thing else in the house; that he put the child into the crib, and that it began to vomit; that its mother came down stairs,—he said the child was then throwing up something, and he went for a doctor. The prisoner was convicted, and executed. The reader will here perceive that the administration was clearly brought home to the prisoner by this well-known effect of the mineral acids in producing symptoms *immediately.* Had the poison been arsenic, the proof of administration would have failed; because this poison may give rise to symptoms within a very variable period, sufficient to have included one or more of the meals which the child had made in the course of the evening.

Page 164. *Retention of volition and consciousness in poisoning by sulphuric acid.*—Mr. Porter mentions the case of a girl, who, after having swallowed a quantity of concentrated sulphuric acid, sat quietly down to tea with some friends, although the quantity of acid taken, was so great as to cause death in a few hours. Another case is related, in which a man took a second dose of the same acid, because he thought the first might not be sufficient. (Med.-Chir. Rev., vol. xxviii. p. 399.)

Page 166. *On the action of concentrated sulphuric acid on the mouth* — In the text it is stated, in reference to the case of *Thomas*, that the fact of there being no marks of injury in the cavity of the mouth, is no proof that sulphuric acid has not been swallowed. The counsel for the prisoner contended that a poison so strong and disagreeable could not be got down the throat of an infant, without leaving *some ill-effect behind in the mouth.* The jury believed this statement, and returned a verdict of acquittal! In addition to the French case, quoted at page 164, which of itself proves that the assumption is untrue and contrary to medical experience, it may be proper to direct the attention of the reader to another, a report of which has been published by Dr. Chowne since this part of the work was printed off. A woman, æt. 52, swallowed from a cup, about a tablespoonful of stong sulphuric acid. She informed Dr. Chowne that she was "strangled" the *moment* it got into her throat, and she fell to the ground. The usual symptoms appeared, and she died in two days. "*The mucous membrane of the cheeks, gums, and tongue, was not excoriated at any part.*" (Lancet, July 10, 1847, p. 36.) It will be seen from this statement that even *concentrated* sulphuric acid may pass through the mouth without necessarily leaving "any traces of unfairness;" and yet the absence of these marks in *Thoma's* case, was actually taken as a proof, not only that sulphuric acid (although diluted) had not been swallowed, but that the corrosion of the œsophagus, stomach, and intestines depended on *natural* causes!

The case related by Dr. Chowne establishes also another important fact, namely, that symptoms are *immediately* perceived, although the mouth may not be chemically corroded by the poison. The woman felt strangled so soon as the acid had reached her throat.

Page 210. *Recovery from large doses of oxalic acid.*—Another case has been lately published by Dr. Barham, in which a woman, æt. 37, swallowed an ounce of oxalic acid, and recovered in about eleven days. Ten minutes after taking the poison she vomited copiously. There was coldness of the surface, followed by tenderness of the abdomen, frequent vomiting and delirium; but the whole of these symptoms disappeared gradually. (Prov. Med. Jour. Oct. 6, 1847, p. 544.)

Page 221. *Poisoning by vinegar.*—A case has been recently published by Dr. David, of Montreal, in which it is alleged a woman swallowed a quart of common vinegar with suicidal intention. When seen, three hours afterwards, she was covered with a cold perspiration; trembling from head to foot; countenance wild; pupils dilated; breathing hurried and laborious; pulse 96, and full; abdomen much distended, and great pain at the scrobiculus cordis. She had no pain or constriction in the throat or fauces, but suffered from intense thirst,—to relieve which she had drunk large quantities of cold water. There were slight efforts at vomiting: she was unconscious of all around her. An emetic of sulphate of zinc was given, followed by carbonate of magnesia. The liquid ejected from the stomach, smelt strongly of vinegar. In about six hours the symptoms had abated, and the woman ultimately did well. (Dublin Med. Press, Oct. 13, 1847, p. 234.)

Page 231. *Doses of the iodide of potassium.*—This salt has been given in very large doses in cases of syphilis, without producing any serious consequences. M. Payen has prescribed as much as sixty grains a day, while Ricord has carried the dose to one hundred and thirty-five grains a day. Some information on this subject will be found in the British-American Journal, July 1847, page 69.

Page 259. *Is arsenic an accumulative poison?*—A case has been lately published by Dr. Hooper, which is of some interest in reference to this question. (Med. Times, Aug. 21, 1847.) A gentleman, æt. 54, afflicted with palsy, was recommended by his physician to take arsenic medicinally. He took five minims three times a day, and continued this practice from October to the 24th of the following June, when he consulted Dr. Hooper respecting an attack of ophthalmia. He was at once ordered to discontinue the arsenic. Dr. Hooper calculated that in eight months and a half this gentleman had swallowed in small doses no less than *sixty-four grains* of arsenious acid! *i. e.* enough to kill thirty adult persons. If arsenic were really possessed of accumulative properties, it does not appear probable that the patient would have survived for so long a period. The poison was probably eliminated so rapidly, that but a small quantity was retained at any time within the system. It was quite sufficient, however, to produce that peculiar train of symptoms indicative of *chronic* poisoning by arsenic:—*i. e.* headach, drowsiness, nausea; an inexpressible feeling of languor and prostration; pulse 90 to 100; increased paralysis; muscular tremors affecting the lower jaw, back, neck, and arms; ophthalmia, accompanied by œdema of the conjunctivæ and erythema of the face; want of sleep, and irritation of the fauces, larynx, and trachea. Under these symptoms the patient gradually sank, and died on the 13th September.

We have here all the characters of slow poisoning by arsenic, indicated by wasting fever and general derangement of the bodily functions. The effects of the Aqua Toffana were very similar to these. Individuals to whom this liquid was administered died without the slightest suspicion of the cause of death being excited. The case proves, what was disputed in that of *Lacoste*, —*i. e.* that arsenic, given in small doses, does not accumulate in the system, and then, without any increase of dose, suddenly give rise to all the well marked symptoms of *acute* poisoning. An occurrence of violent symptoms of alvine irritation would always justify the inference that another and a larger dose had been taken shortly before. It would be advisable in these cases to collect the urine of the patient for several successive days, and then submit it to analysis. This might be easily done, and a diagnosis obtained without any suspicion being excited in the mind of the administrator.

Page 277. *The ammonio-sulphate of copper as a test for arsenic.*—The various methods of identifying the precipitate as *arsenite* of copper have been fully explained in the text. It has been lately announced by Dr. Cattell, (Lancet, Oct. 9, 1847, p. 386,) "that prussiate of potash constitutes an excellent test for arsenite of copper, with which it strikes a blood-red colour. This statement was also publicly made at an inquest lately held in Cambridgeshire. According to my observation, the prussiate of potash is a test for *oxide of copper*, and not for *arsenite* of copper. Thus the subchloride, the subacetate, and other insoluble salts of copper are equally turned red by the supposed test. Hence it cannot enable us to distinguish the arsenite from the other insoluble salts of copper.

Page 283. *Deposits of arsenic and antimony.*—In addition to the process described in the text, it may be proper to state that various other methods have been proposed to distinguish arsenical from antimonial deposits obtained by Marsh's apparatus. This is the more necessary, since at some recent trials, questions on the subject have been put to scientific witnesses. The following is an extract from the evidence of Mr. H. H. Watson in the case of the *Queen* v. *Johnson*, Liverpool Lent Assizes, 1847. The examination turned upon the detection of arsenic in the tissues by Marsh's process:—"I tried experiments with the metallic deposits, by applying chloride of lime to those obtained by Marsh's process on glass and porcelain, which removed them. That, together with the other results, indicates that the metallic deposit is not antimony, and that it is arsenic. From my own experience and knowledge I should say it was arsenic, though I cannot say chloride of lime will not remove other substances besides arsenic. It will not remove antimony. I also exposed some of these metallic deposits on glass to a temperature ranging from 355 to 565 degrees, by which they were volatilized and left the glass. This is another proof that the metal is not antimony, but arsenic; antimony does not volatilize at the temperature mentioned, but remains permanent, while it is one of the properties of arsenic to become volatilized at that temperature. Some of the slips of copper on which I had got a strong steel-like metallic deposit by Reinsch's test, were exposed to heat in a glass tube:—the metallic deposit became oxidized, and deposited itself higher up in the tube. That oxide I afterwards boiled in some distilled water, and tried again in the ammoniacal nitrate of silver, ammoniacal sulphate of copper, and sulphuretted hydrogen, and they gave the same precipitates as before, from which I concluded that the water contained arsenious acid. I have no doubt that the body I saw exhumed, contained arsenic. I cannot say how much—perhaps not more than a grain, if so much, in the nineteen ounces of intestines I operated upon." There can be no doubt from this statement that arsenic was clearly and satisfactorily detected. In reference to this evidence on the distinction between arsenic and antimony, the learned judge made the following remarks in his charge to the jury: "the application of chloride of lime proved that the metallic substance found in the body was not antimony because it vanished, but it did not prove that it was arsenic, *because that test might apply to some other substance.* It is a negative test as to antimony; but it is an affirmative test as to arsenic *or some other metallic substance.*" I would beg to observe that there is here a complete misapprehension as to the application of the test. The real point to be considered, is not as represented by his lordship, what other metallic substance besides arsenic will or will not be dissolved by chloride of lime, but what other metallic substances there are *besides antimony* which will combine with hydrogen to form an inflammable gas and become deposited on glass in a form to be mistaken for arsenic! The object of the analyst is to draw a clear distinction between these *two metals*, which alone are deposited, and are likely to be confounded in the preliminary process. He may hold himself perfectly indifferent as to the effects of chloride of lime, or any of the processes used for this distinction, upon all other bodies in chemistry. When it is proved that there is any metal besides arsenic and antimony, which can be thus deposited on glass from Marsh's apparatus, it will then be time to consider how far the process employed may be safely trusted. This mode of placing the facts before the jury, could lead to no other inference than that the substance which was conclusively proved to be arsenic, (by the use of chloride of lime, *taken together with its mode of deposition*) was *not arsenic*, but that it might have been "some other metallic substance." Thus the evidence of the presence of the poison, which to all chemists must be clear and satisfactory, was placed under a degree of doubt which a proper and scientific view of the facts does not warrant!

M. Cottereau gives the following summary of the numerous processes which are open to the analyst for making a chemical distinction between these deposits. (Journal de Chimie, 1846, 330.)

1. The action of *Heat*, which volatilizes the arsenical stain with a garlic odour, while the antimonial stain is fixed. [This is not a satisfactory method when the deposits are small.]

2. The action of *Chloride* (hypochlorite) *of soda*, or chloride of lime. This dissolves the arsenical but leaves unchanged the antimonial deposit. This method was first suggested by Bischoff.

3. The action of *cold nitric acid*, and the application of nitrate of silver or sulphuretted hydrogen gas to the residue obtained by evaporating the acid liquid to dryness. [This is the plan recommended in the text, nitromuriatic being substituted for nitric acid; and nitrate of silver being employed as the test. The operation may be completed in less than three minutes, and the results are free from any chemical objection.

4. The action of *Iodine vapour*, which causes the arsenical deposit to disappear, the iodide of *arsenic* being subsequently indicated by a deep yellow colour from the action of a current of sulphuretted hydrogen gas upon the residue. [This method was first proposed by Lassaigne.] The experiment may be conveniently performed by inverting a watch-glass, holding the suspected stain, over another containing a few grains of iodine. The vapour at common temperatures produces with arsenic a deep citron-yellow coloured compound, iodide of arsenic,—while the antimonial stain under similar circumstances, is turned of a deep orange or vermilion red colour. It has been

further suggested by Lassaigne that hydriodic acid containing iodine, or an alcoholic solution of iodine, immediately dissolves the arsenical stain, leaving a yellow deposit on evaporation, while with antimony there is no immediate action,—the stain is slowly dissolved, and leaves, by evaporation, a vermilion red coloured residue. (Journal de Chimie, 1846, p. 16.) According to my experiments this process is unsatisfactory: the results are slow, and they have after all only a relative value.

5. The action of *Hydrosulphuret of ammonia*, which dissolves the antimonial deposit and often detaches that of arsenic in scales. [This is a useful test, because it has a direct action on antimony.] Dr. Guy has recently suggested that the mixture should be evaporated, when, if the stain be antimony, the residue will be orange-red: if arsenic, yellow. (Med. Times, July 17, 1847, 411.) Care must be taken not to use an alkaline persulphuret; for the excess of sulphur left on evaporation gives a yellow colour with an antimonial stain. In employing this process it is always advisable to test the coloured residue. The sesquisulphuret of antimony is dissolved by strong muriatic acid, and not by ammonia,—that of arsenic by ammonia and not by muriatic acid. (See pages 278, 393.) Further, the muriatic solution of antimony added to water produces, under proper precautions, a white precipitate of subchloride of antimony. (See page 395.)

6. The action of *Chlorine*, which causes the arsenical stain to disappear,—the deposit being again rendered yellow by a current of sulphuretted hydrogen. [This is not satisfactory, because the chlorine of the chloride of arsenic may cause a separation of sulphur.]

7. M. Cottereau recommends, in preference to all these methods, the exposure of the stain to the vapour of *Phosphorus*. This may be done by cutting the phosphorus in small pieces and employing it like iodine, (See 4, supra.) In a few hours the arsenical stain entirely vanishes,—that of antimony remains. Heat accelerates the action, and arsenic may be detected in the residue by a current of sulphuretted hydrogen gas. [This difference of action here described is very apparent, but it requires some hours.]

8. M. Boutigny advises that the following points should be successively observed in order to identify an arsenical stain:—1, its iron-grey lustre; 2, its solution in diluted nitric acid by heat; 3, the formation of a yellow sulphuret by sulphuretted hydrogen; 4, by this sulphuret forming a colourless solution with ammonia; 5, by its being again turned yellow on touching it with muriatic acid. This experiment may be performed by throwing the ammoniacal solution on a red-hot platina capsule, so as to produce the spheroidal condition; 6, the alliaceous odour. (Journal de Chimie, 1846, page 10, and page 446.)

For reasons already assigned, the process which appears to me preferable for simplicity, rapidity, and certainty, is that described in the text (p. 394, 282,) and under 3. It answers as well when the analyst has to deal with a mixed stain of arsenic and antimony, (a difficulty left untouched in most of the above methods,) as with a stain of either metal separately.

Page 293. *Orfila's new process for arsenic in tissues.*—The method lately proposed by Orfila consists in bruising up about eight ounces of the liver, or other organ, with a sufficient quantity of water to make the whole perfectly liquid. A strong current of chlorine gas is then passed into the liquid, for seven or eight hours. A kind of frothy coagulum of animal matter is thereby formed: this is allowed to subside, and in about twelve hours the supernatant liquid is filtered off, boiled to expel the excess of chlorine, and then examined by Marsh's apparatus. If well-marked arsenical deposits be not procured, the solid residue is decomposed by boiling with strong nitric acid, and the whole of the arsenic is then obtained in the evaporated residue, as arsenic acid: this may be dissolved in distilled water and tested. One-third of the liver of the *Duke of Praslin* gave, by this method, an abundance of arsenic.

In the official analysis in the case of the Duke of Praslin, this process was pursued; but concentrated and pure sulphuric acid was employed to destroy the chlorinated organic matter, instead of nitric acid, as recommended by Orfila. It was remarked that the addition of sulphuric acid caused an abundant evolution of muriatic acid vapour and of sulphuretted hydrogen gas. (Ann. d'Hyg. Oct. 1847, 404.)

I find it very difficult to reduce the liver to a pulp with water; but by cutting the organ into fine pieces, the chlorine converts the whole into a soft, white, pasty mass, and fixes the arsenic at once as arsenic acid. This appears to me to be the chief advantage of the new plan.

Page 298. *Arsenic in the earth of cemeteries.*—The earth of the churchyard in which *Johnson* was buried has, since the remarks in the text were written, been analysed by Mr. Watson. This gentleman informs me that he procured a pound of earth at the depth of two yards (the depth of Johnson's grave,) and at the distance of two and a half yards from the foot of the grave, and submitted it to analysis by a process similar to that described. The result was that *no trace, of arsenic,* either in a soluble or insoluble form, was discovered. The prisoner appears to have been acquitted chiefly on the assumption that the earth did not only contain arsenic, but that this arsenic was in a state to be dissolved by rain water, and washed into the body! A few questions put to the witness who made the analysis, would have shown that this was, under the circumstances, an inadmissible assumption. Although the evidence of Mr. Watson was so materially affected by the result, no questions were put to him on the subject either by the learned judge or counsel. The quantity of arsenic found in the body was small, but *absorbed* arsenic is never found excepting in small quantity (see p. 117;) and in Johnson's case, as in all others, the analysis was limited to the clear demonstration of the *presence* of the poison. A portion of the soft parts (nineteen ounces) only was examined; and it cannot be regarded as indispensable to chemical evidence, that every molecule of poison contained in every organ and tissue of the body, should be extracted on these occasions. The soft tissues *not* examined, in the case of Johnson, probably contained just the some proportion of arsenic as those which were analysed; although the Court appears to have adopted the view that there was no more arsenic in the whole body than that produced from less than a pound and a half of the viscera!

Arsenic is contained in nearly all specimens of brass and iron and when nails of these metals are used for coffins, it would be just as reasonable to assume that the poison found in an exhumed body, was derived from them, as, under the circumstances proved in this case, that it was derived from the earth around the grave. In reference to Mr. Watson's exclusion from Court, I have been since informed by this gentleman, that before the trial, it was ordered that no witness should be allowed to be in Court until called for; and as no special exception was made in his favour, he did not think himself justified in entering.

Page 310. *Poisoning by the vapour of mercury.*—Although metallic mercury is not poisonous, yet when it is respired in the state of fine vapour, it enters the body through the lungs, and is then capable of producing serious symptoms, and even death. Mercury may pass into vapour at all temperatures:—at about 80° it is rapidly diffused in the most finely divided state, a fact illustrated in Daguerreotype art by its deposition on polished silver. In all the trades in which mercury is handled, it may thus penetrate into the system by slow degrees. The chronic effects are manifested by tremors and paralysis of the limbs,—a state called shaking palsy,—vertigo, loss of memory, disturbance of the intellectual faculties, salivation and ulceration of the mouth, colic, general emaciation, and death. A blue line, as in chronic poisoning by lead, may be found around the edges of the gums. Water-gilders, and the manufacturers of looking-glasses, barometers, and thermometers, are very subject to these disorders. The frequent contact of mercury with the hands, may suffice to produce them in a modified degree. A singular medico-legal case, in reference to the noxious effects of mercurial vapour, is reported by M. Chevallier. (Ann. d'Hyg. 1841, i. 389.) It was alleged, in this case, that two children had suffered seriously in health, in consequence of the distillation of mercury being carried on in an apartment below that in which they lived. They had general tremors and other symptoms indicative of mercurial action; but there was no salivation. It has been remarked that those who are subject to the shaking palsy are not very liable to become salivated. M. Chevallier detected mercury in the dust of the apartments on all the floors of the house; and his conclusion was, that the disordered health of the children was certainly due to these mercurial emanations.

One of the most remarkable instances of the noxious effects of mecurial vapour, was observed in the case of the *Triumph,* while conveying a cargo of quicksilver off Cadiz, in April 1809. By some accident the leathern bags containing the metal burst, and *three tons* of quicksilver were dispersed through the vessel. The crew soon began to suffer from salivation, partial paralysis, and disorders of the bowels. In three weeks no less than two hundred men were salivated. Two men died from excessive salivation: one had previously lost his teeth, and his cheeks were in a gangrenous condition: the other had also lost the whole of his teeth, the greater part of his tongue, and at the time of his death the lower lip was in a state of gangrene. The interior of the ship was covered with a black powder, and the copper bolts were mercurialized. The vapour proved fatal to the animals on board; for nearly all the poultry, sheep, pigs, mice, goats, cats and dogs, and even a canary bird, died from its influence. (Paris, Med. Jur. ii. 461.)

Great danger is always to be apprehended when any operations with metallic mercury are carried on in small and ill-ventilated apartments, heated to a temperature above 70°. The best test for the detection of these vapours, is the suspension of a piece of pure gold leaf in the apartment. If mercury be present this will become slowly whitened by amalgamation.

Page 352. *Iodide of potassium as a test for lead.*—A statement has been lately circulated in some of the medical journals, that this salt is an unsatisfactory test for lead, because it has the property of redissolving the yellow iodide of lead. The facts stated in the text, will show that it will without difficulty detect a very small portion of acetate of lead; and at page 354, a case is mentioned, where minute traces of lead were easily detected in the incinerated liver by the aid of this test. Mr. Herapath, Jun. states, that a small quantity of acetic acid added to the liquid, increases its power as a reagent. I do not find that it possesses any perceptibly solvent action on the precipitate in neutral solutions at common temperature; but if the iodide of potassium contain any potash or carbonate of potash, or if the salt of lead be diffused through a large quantity of water, or dissolved in strong muriatic acid, the precipitate will either not be produced, or it will be formed and immediately redissolved. The iodide of lead is to a certain extent soluble in boiling water, and even when it is not entirely dissolved, its yellow colour is destroyed at 212°.

Page 361. *Blueness of the gums in poisoning by lead.*—In the text it is stated that mercurial preparations cause this appearance at the edges of the gums. Dr. Branson, of Sheffield, has observed a similar blue line in patients taking *Nitrate of silver;* and he considers it a fair criterion of the introduction of this metal into the system, so as to render it a means of avoiding discolouration of the skin.

Page 389. *Fatal effects of tartar emetic upon young children.*—The facts mentioned in the text, as resulting from the experience of Mr. Noble and Mr.

Goodlad, have been recently confirmed by the observations of French practitioners. Three quarters of a grain of tartar-emetic were prescribed for a slight gastric disorder, in the case of an infant recovering from measles. The child died in an hour. A similar dose was prescribed for another child of the same parents: violent vomiting and purging supervened, and this case also ended fatally. In a third instance of a girl, æt. 4, attacked with hooping cough, one-third of a grain given in divided doses, produced very alarming effects, which only disappeared by discontinuing the medicine. In a fourth case, the recovery of the child from a dose of tartar-emetic, was followed by desquamation of the cuticle. (Journal de Chimie, Septembre 1847, 471.) It is unfortunate that the particulars of these cases are not more fully given.

Page 425. *Recovery from a large dose of Croton oil.*—Dr. Cowan quotes the following case of poisoning by croton oil in his Retrospective address:—A teaspoonful was administered by mistake to a child four years old, who had previously eaten a hearty meal of bread and milk. In five minutes the child was seized with violent vomiting and purging, soon followed by alarming prostration. Under the use of warm fomentations, and free libations of milk and mucilage, the child was convalescent in two days. (Prov. Trans. N. S. i. 121.) The recovery was here probably due to the oil having been taken on a full stomach, and to early vomiting. Dr. Cowan states, that he has known similar symptoms follow the administration of half a drop to an adult.

Page 429. *Poisoned bread.*—A case is mentioned in the Gazette Médicale, (4 Septembre, 1847,) in which a large quantity of brown bread, baked two days previously, spontaneously acquired unwholesome properties by the rapid growths in it of a peculiar kind of fungus—the *Oidium Aurantiacum.* The unwholesome portions were indicated by large white patches contrasting strongly with the brown colour. These patches had the nauseous odour of poisonous fungi, and became yellow when heated to about 114°. The appearance of the bread led to its being rejected as food.

Page 437. *Post-mortem appearances in poisoning by cantharides.*—A lunatic, æt. 45, swallowed by mistake half an ounce of cantharides-plaster containing two drachms of the powder. Remedies were immediately applied. In about two hours the whole of the mucous membrane of the mouth was red, and covered with small blisters. In seven hours there was great coldness of the surface, with imperceptible pulse. The urine passed was mixed with blood. Death took place in twenty-four hours. On inspection, the vessels of the brain were found gorged with blood, and a quantity of serum was effused in the ventricles and between the convolutions. The heart and lungs were healthy. The internal surface of the stomach was covered with red points interspersed with ecchymosis, in the centre of which was seen an adhering particle of the powder of cantharides. The intestines were healthy, but the kidneys were red and gorged with blood. The left ureter internally was of a very red colour. The bladder was thickened, and the mucous coat injected with blood. (Ed. M. and S. J. Oct. 1844, p. 563.)

Page 457. *Hydrophobia from disease.*—Hydrophobia is a symptom which is occasionally met with in certain cases where there can be no suspicion of the individual having been inoculated by the poison of rabies. Mr. Stafford describes a case of tetanus from an accidental injury to the foot, in which hydrophobia was one of the most prominent symptoms. The case speedily proved fatal. (Med. Gaz. xxxv. 827.)

Incubation of the poison of rabies.—In the text it is stated that the longest ascertained period at which hydrophobia has appeared after the bite of a dog was twelve months. Mr. Allan has since published an interesting report of a fatal case, in which the symptoms did not manifest themselves until *thirteen months* after the bite. (Lancet, Oct. 16, 1847, 409.)

Page 478. *Symptoms resembling narcotic poisoning in disease of the kidneys.*—Mr. Corfe has reported a case in which the symptoms of diseased kidney were strikingly similar to those caused by opium. The female was in a state of perfect insensibility, which had existed for ten hours prior to her admission into the Middlesex Hospital: the pupils were *contracted*, and there was convulsive slow breathing. She died in four hours and a half. There was strong presumptive evidence of poisoning, as a three-ounce phial, containing laudanum, was seen in her possession the preceding day, and could not then be found. It was, however, proved at the inquest that the laudanum had been used by another person as a liniment, and that the deceased could not have taken any portion of it. The inspection showed that death was clearly due to granular degeneration of the kidney; and on inquiry it was ascertained that the urine, for some months previously, had been strongly albuminous. (Med. Times, Oct. 9. 1847, 648.) This case conveys an important lesson on diagnosis in narcotic poisoning.

Page 499. *Death from the external application of opium.*—A soldier æt. 32, was attacked with phlegmonous erysipelas on the face and outer part of the right leg, on account of which a linseed poultice, moistened with *fifteen* drops of laudanum, was applied to the limb. Next morning he was found in a state of deep sopor: the eyelids tremulous and half open, pupils contracted, the lips distorted, the muscles of the face affected with spasm, and those of the limbs with convulsions. The medical attendant, perceiving a strong odour of opium, examined the bandages, and found them soaked with laudanum—the hospital servant having ignorantly applied nearly an ounce! In spite of treatment the convulsions increased,—the pulse became more feeble and the patient died. On inspection some red points were seen on the arachnoid: a strong opiate odour was exhaled from all parts of the body, and the heart, stomach, and brain, were healthy. None of the poison could be detected in the blood. (Ed. M. and S. J., xxix. 450; and Journal de Chimie Médicale, Avril 1827.) The application of opium in any form to an abraded surface of skin, is liable to give rise to all the results of narcotic poisoning.

Page 518. *Effects of prussic acid vapour.*—Since the remarks were written, a case of poisoning has occurred in this metropolis (Oct. 29, 1847.) involving the question whether the vapour of Scheele's acid, if respired, would destroy life. The deceased entered a druggist's shop, and requested to be shown a bottle of Scheele's prussic acid. He suddenly attempted to snatch the bottle from the hand of the assistant; a struggle ensued, during which a portion of the acid was spilled over the deceased's face, and over the coat of the assistant. The deceased ran into a neighbouring shop, and died in about a quarter of an hour. At the inquest it was alleged that death had been caused by the vapour, owing to the acid having been spilled over the deceased's face. Of this, however, there was no proof, as the body was not inspected for the inquest! The same question arose in a case of poisoning by the essential oil of almonds (ante, p. 572;) but in that instance the suspicion was shown to be unfounded, by the discovery of the poison in the stomach. It is most probable that a sufficient quantity of the acid to cause death was in this instance swallowed.

Page 527. *External application of prussic acid.*—The facts mentioned in the text, will suggest a caution in reference to the medicinal employment of prussic acid locally. It is sometimes used, and with good effect, to allay the violent itching which attends certain diseases of the skin. It has also been applied to the os and cervix uteri for the alleviation of pain. As, in the latter case, it meets a highly absorbing surface, care should be taken so to regulate the proportion of the acid, both in absolute quantity and degree of dilution, as to prevent the possibility of danger. Dilution does not appear to make so much difference as absolute quantity, and this may be regulated by the known fatal dose. (See page 538.)

Page 545. *The odour of prussic acid in the stomach. Tawell's case.*—As a casual statement, made by Orfila, was represented to imply that the odour of bitter almonds (prussic acid) was present in the cases of the Parisian epileptics, and that, in short, the odour was never absent when a person had died from the effects of the poison, I here quote Orfila's opinion on this subject, from his report of the case of M. Pralet:—"Quelle foi peut on ajouter au caractère tiré de l'odeur d'amandes amères, qu'aurait répandu le cadavre du Sieur Pralet (point sur lequel on est loin d'être d'accord,) lorsque nous voyons MM. Marc, Marjolin et Adelon déclarer, que nulle partie des cadaveres des épileptiques de Bicêtre n'exhalait l'odeur d'amandes amères; qu'il en était de même chez l'éleve en pharmacie dont j'ai parlé; et que nous savons à ne pas en douter, qu'il *n'est pas rare de ne pas reconnaître cette odeur* dans les organes des animaux empoisonnés par l'acide cyanhydrique?" (Ann. d'Hyg. 1841, ii. 409.) It is here circumstantially stated, upon authority which Orfila must have considered better than his own, that no odour was perceptible in the bodies of the epileptics; that none was perceived in another case to which he refers; and that it was by no means unusual to find the bodies of animals, poisoned by the acid, free from any odour. Coupled with the original report made by Orfila in 1829, this must be taken as conclusive of the unfairness of the imputation thrown upon English writers, that they had misstated the facts, and misrepresented Orfila's opinion.

Page 557. *Alleged spontaneous production of prussic acid.*—Since the remarks in the text were written, Dr. Guérard has published a translation of a paper by me on the sulphur-test for prussic acid, and appended to it a note in reference to the spontaneous formation of prussic acid in the human stomach. (Ann. d'Hyg. Oct. 1847, 442.) He states that M. Filhol has recently ascertained by experiment, that nearly all vegetable substances containing nitrogen, possess the property of transforming *amygdaline* to essential oil of bitter almonds and prussic acid. A mixture of common flour, barley, or rye, produces this effect in *five or six hours;* but with maize, it required a period of three or four days. He then inquires whether the stomach might not possibly contain the materials which would lead to this transformation; and in the event of sudden death from natural causes, might there not be a false accusation of poisoning by prussic acid?

If the human stomach contained any substance taken as *food* or medicine, from which prussic acid was producible, such a mistake might occur; but M. Filhol's experiments show that *amygdaline* must be present; and this peculiar body is not a constituent of food. It is found in the bitter almond, in peach kernels, and in the berries and leaves of the laurel. If these substances have not been eaten with the food, or not taken in sufficient quantity to account for death, no mistake can arise: if, however, they have been taken, then the case resolves itself into one of poisoning by these substances. If the individual has swallowed pure amygdaline, the same result would happen; but the real force of this objection to chemical evidence, is not to produce prussic acid from amygdaline, but to produce amygdaline from ordinary food or by common putrefactive fermentation. It is as easy to conceive that prussic acid should be formed in the stomach at once from beef, potatoes, and other substances, as that amygdaline should be derived from them. It may be proper to state that sweet almonds and common nuts contain no amygdaline; its presence is indicated by the powerful odour of the essential oil on trituration with water.

Page 567. *The quantity of prussic acid obtained from the bitter almond.*—This became a material question in a case of suicide which occurred in this metropolis in November 1844. A man was found dead, and there was a strong odour of bitter almonds in the room in which his body was discovered: there was also the appearance of froth about the mouth of the deceased. On inspection, some bitter almonds in an undigested state were found in the stomach. The quantity of prussic acid obtained by a distillation of the contents, was stated to be equal to one drachm of the common acid (P. L.) The medical practitioner is reported to have said, that this acid might not have been taken as such, but derived from the almonds. The anhydrous prussic acid obtained, amounted to 1·2 grains, *i. e.* quite enough to destroy life. The quantity of bitter almonds required to yield this, according to the calculation in the text, would be about 500 grains. The weight of the almonds found in the stomach is not stated; but it is not likely that so many could have been eaten as to produce and leave in the stomach this large quantity of anhydrous prussic acid, nor would this account for the odour in the apartment. It is highly probable that the individual destroyed himself with prussic acid, having previously eaten some bitter almonds in order to conceal the odour of the poison.

Page 618. *Poisoning by belladonna.*—MM. Bayard and Chevallier have recently reported several interesting cases of poisoning by belladonna. Two persons swallowed a small spoonful of the *extract* by mistake for that of juniper. There was speedily indistinctness of vision, tottering gait, delirium, incoherency, hallucinations, and dilatation of the pupils. In one there was a great deal of cerebral excitement. The apothecary to whom the extract was taken, tasted it, and soon experienced symptoms which led to a suspicion of its real nature. Under treatment the symptoms of poisoning disappeared in two days; but one of the patients died on the seventh day, from phlebitis, complicated with a generally diseased condition of the body. The physical and physiological properties of the extract indicated that it was belladonna; but the attempt to procure atropia entirely failed. A portion of the concentrated extract given to a dog caused dilatation of the pupil *in a quarter of an hour*,—an index of the rapidity with which atropia is absorbed. In a second case a young man swallowed an infusion of two drachms of the *leaves* of belladonna. In about an hour he found great difficulty in swallowing, the salivary secretion was suppressed, and objects appeared to be in perpetual motion before him. He became delirious; attempted repeatedly to pass his urine, but could not; and for an hour and a half he was in constant motion, although his gait was unsteady. The muscles of his face, jaws, and limbs were agitated by convulsive twitchings; the pupils were excessively dilated, and there were singular hallucinations. There was neither nausea, vomiting, nor diarrhœa. Emetics, enemata, and venesection were resorted to; and the next morning he awoke as if from a dream, complaining of a feeling of lassitude.

A woman swallowed, on an empty stomach, a *drachm* of the extract. She then took some food. No symptoms appeared for *three hours:* they then came on suddenly. When seen an hour afterwards, she could not stand; there was trembling of the limbs, with convulsive motions; a nervous laugh and incoherent speech. The pupils were much dilated, and great lassitude followed this stage of excitement. Under treatment she recovered in twelve hours. (Ann. d'Hyg Oct. 1847, 413.)

Atropia.—The reader will perceive that I have mentioned no tests for the presence of this alkaloid. It is described in some chemical works as yielding a white precipitate with tannin, and yellow precipitates with the chlorides of gold and platina. These, however, have no pretension to the name of *tests*. Tannin precipitates the solutions of all the alkaloids, and the chlorides of gold and platina give yellow precipitates with the solutions of strychnia: hence, there is nothing peculiar in their action upon atropia. The only indication of the presence of atropia is that derived from the action of its solutions in causing dilatation of the pupil; but this property is by no means peculiar to it.

We are still in want not only of simple processes for the separation of most of the alkaloidal poisons, but also of chemical *tests* to determine their nature when separated. It has been too much the practice with respect to these poisons, to assume that any reagent which causes a precipitate, or change of colour, becomes a test: but there cannot be a greater fallacy. So long as such speculations are confined to books, this mistake is immaterial: but if any attempt be made to pass these casual results as *proofs* of the presence of the poison, in a Court of Law, their insufficiency will no doubt be speedily exposed in the cross-examination.

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ERRATUM.

Page 284, line 16, *for* "one-thirteenth of a grain," *read* "the one hundred and thirtieth part of a grain."

THE END.

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